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CARDIAC ARRHYTHMIAS.[1]

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No apology is needed for the title of the subject on which I have the honour of addressing you this evening, as the elucidation of the various types of cardiac arrhythmia forms a considerable part of the general practitioner's routine work. My purpose is to discuss these various irregularities of the heart beat, to throw some light on their ætiology and diagnosis, and to suggest appropriate treatment.

In the first place, we may with advantage review our knowledge of the origin and propagation of the contraction wave in the heart. The impulse to contraction arises in the sino-auricular node, an ill-defined neuro-muscular mass of tissue situated in the *sulcus terminalis* near the entrance of the *vena cava superior* into the right auricle.

It was first described by Keith and Flack in 1907, and since confirmed by Sir Thomas Lewis, Oppenheimer and others. This node receives its blood supply from a small artery which springs from the right circumflex artery; in human beings it not infrequently anastomoses with a corresponding branch from the left coronary artery. The sino-auricular node has also nervous connexions with the vagus and the sympathetic.

The wave of contraction commences here in the sino-auricular node, or pace-maker, it then passes down through the auricular musculature and it is propagated from one muscle fibre to another until it reaches the auriculo-ventricular node.

[1] Read at a meeting of the Queensland Branch of the British Medical Association, on October 2, 1931.

All authorities are agreed that there is no special conducting mechanism in the auricles. The connecting link between the auricular and ventricular musculature was first described by Stanley Kent in 1892, and again by His in 1893; and later again Tawara gave a complete account of the auriculo-ventricular node and bundle, and traced its connexions to the network and arborizations of Purkinje.

In this way we may trace the fibres of the junctional tissues from auricle to ventricle. It commences at the base of the auricular septum, near the coronary sinus, where the auricular fibres collect fan-wise and become united with the auriculo-ventricular node, which lies on the postero-inferior surface of the right side of the auricular septum. From here the main bundle passes forwards in a special canal to the right of the *pars membranacea septi* of the ventricles, to divide into right and left bundles. The right bundle passes on down the right side of the septum to the moderator band, and then by this means to the papillary muscles and to the Purkinje network.

The left bundle immediately perforates the septum, between the posterior and right anterior aortic cusps, and emerges on the left side of the septum, to pass on to the papillary muscles and network.

It has been shown by Stannius's classical experiment that, when the sinus is ligated, it beats on at its own rate; the auricle and ventricle, after a short pause, commence to beat at a slower rate. When another ligature is placed between the auricle and ventricle, the auricle goes on beating at its revised rate, and after a short period of asystole the ventricle commences to beat again at an even slower rate.

This experiment shows that: (i) Each chamber of the heart has the power of initiating its own rhythm and is capable of carrying this on when separated from the others; (ii) that there is a descending order in the rhythm rate, the sinus beating fastest, then the auricle, and finally the ventricle; (iii) that the part which has the fastest rhythm becomes the pace-maker of the heart, and overrides the slower rhythms.

Sinus Irregularities.

Sinus irregularities are mostly found in young people, especially children. They are due to vagal action upon the sino-auricular node, and cause the pulse rate to wax and wane with respiratory movements. It quickens during inspiration and slows during expiration. (See Figure I.) On account of its being vagal, this irregularity readily disappears with any increase in the pulse rate. Therefore, it disappears with exercise, in fevers and shortly after the administration of atropine or amyl nitrite.

Severe sinus interruption may lead to complete cessation of the heart-beat and account for syncope in some cases. But the common form is of no clinical importance and may be disregarded. No treatment is necessary.

Extrasystoles or Ectopic Beats.

Mackenzie, by means of his polygraph, was the first to recognize the nature of extrasystoles and to distinguish this form of irregularity from the more serious condition of auricular fibrillation.

Extrasystoles or ectopic beats, as Lewis calls them, occur when some portion of the heart muscle other than the normal pace-maker becomes irritable and initiates a stimulus which produces a contraction. This ectopic focus may be in the auricles, in the auriculo-ventricular node or in the ventricles.

When the extra beat arises in the auricle, the normal rhythm from the sinus only is disturbed, and there is no very apparent compensatory pause. On the other hand, when it arises in the junctional

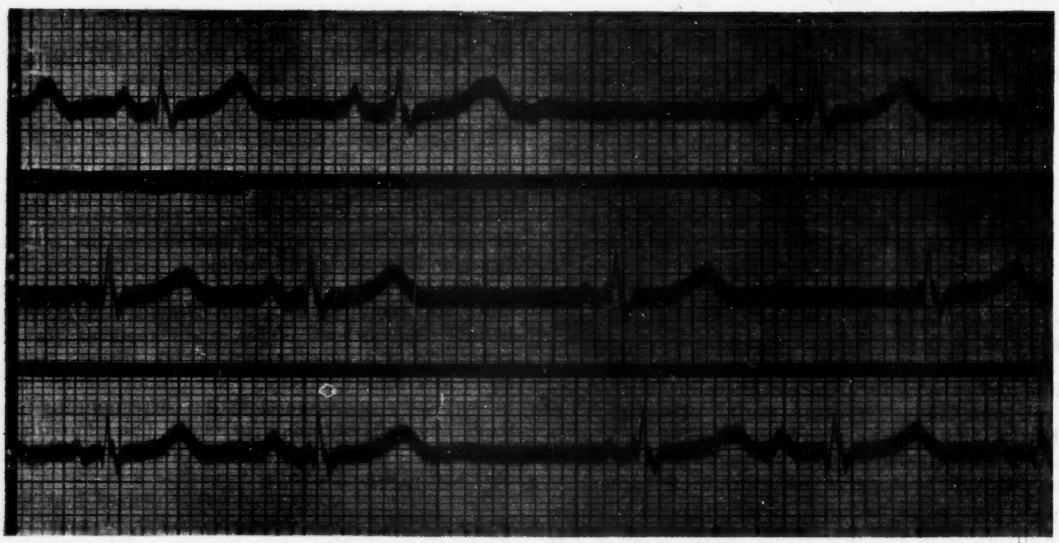

FIGURE I.
Sinus arrhythmia all Lead II in a female patient aged sixty-three years with an enlarged heart and mitral incompetence.

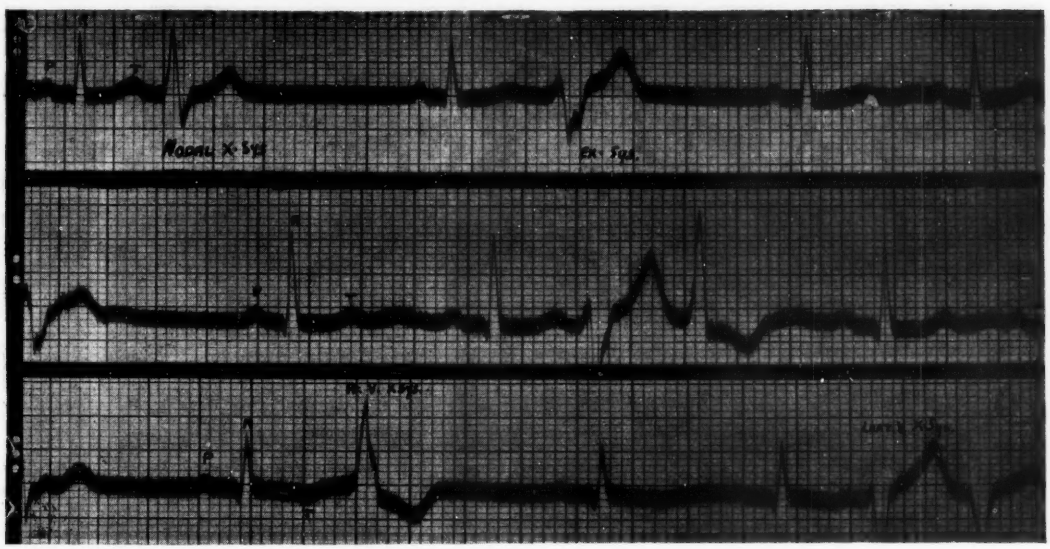

FIGURE II.
Nodal right and left ventricular extrasystoles in a female patient aged nineteen years with subacute rheumatic carditis.

tissues or in the ventricles, the sinus rhythm is not interfered with, and there is a compensatory pause after the premature beat, so that the pauses before and after it are together equal to two normal beats. (See Figure II.)

The electrocardiogram has been of the greatest assistance in the elucidation of these irregularities. The tracing at once indicates the site of origin of the extrasystole, whether in the auricle, the auriculo-ventricular node, or right or left ventricle. When the impulse is supraventricular, that is, commencing in the auricle or node, the *QRS* wave is normal in shape. When it is a ventricular extrasystole, it is quite abnormal and characteristic. The *P* wave may or may not be visible, because of its inclusion in the ventricular complex.

Auricular extrasystoles occur more or less evenly in persons up to sixty-five years of age; nodal extrasystoles are much commoner in old age, and ventricular extrasystoles are most commonly present between the ages of fifteen and thirty, and again between forty and seventy.

Extrasystoles in the younger age group are chiefly due to rheumatic conditions, and those of the older group to arteriosclerosis and arterial hypertension. (See Figure IIIA.)

Maurice Campbell reports the incidence of 20% or 100 cases of extrasystole in the course of electrocardiographic examination of 500 patients at Guy's Hospital. Of this number, there were roughly equal numbers of rheumatic, arteriosclerotic and healthy hearts, with a few associated with goître.

Of the rheumatic patients practically all had an exacerbation of the rheumatic process at the time, and so most probably a rheumatic myocarditis. In the arteriosclerotic group practically all had definite evidence of gross cardiac changes. The third group gave no apparent evidence of heart disease, and some of them who had been followed up to six years did not sustain much damage.

Extrasystoles appear to have little significance when present in apparently healthy hearts, but when there is evidence of cardiac disease, they are of the greatest importance.

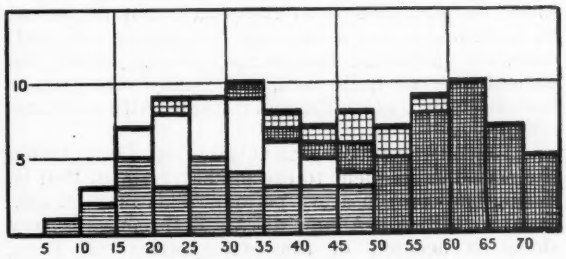

FIGURE IIIA.
The ætiology and age incidence of extrasystoles (all types).
The horizontal shading shows rheumatic cases; the white areas, cases with no apparent heart disease; the heavy cross-shading, cases with high blood pressure or arteriosclerosis; and the light cross-shading, cases with exophthalmic goître.
(Incidence shown as percentage of 100 cases for comparison with Figure IVA.)

In rheumatic persons they point to the presence of active infection or an overworked heart. In exophthalmic goître they point to an active stage of the disease or to the presence of added infection.

In persons with arteriosclerosis they are usually associated with signs of cardiac failure, and in some cases appear to be the forerunner of the onset of failure. (See Figure IIIB.) They may indicate over-digitalization, which usually takes the form of coupled beats.

The presence of extrasystoles may lead to the greatest discomfort and concern. The patient is disturbed by the "thump" which he feels in his chest, he may say that he feels his heart give a jump or turn-over. In some cases the premature beat is

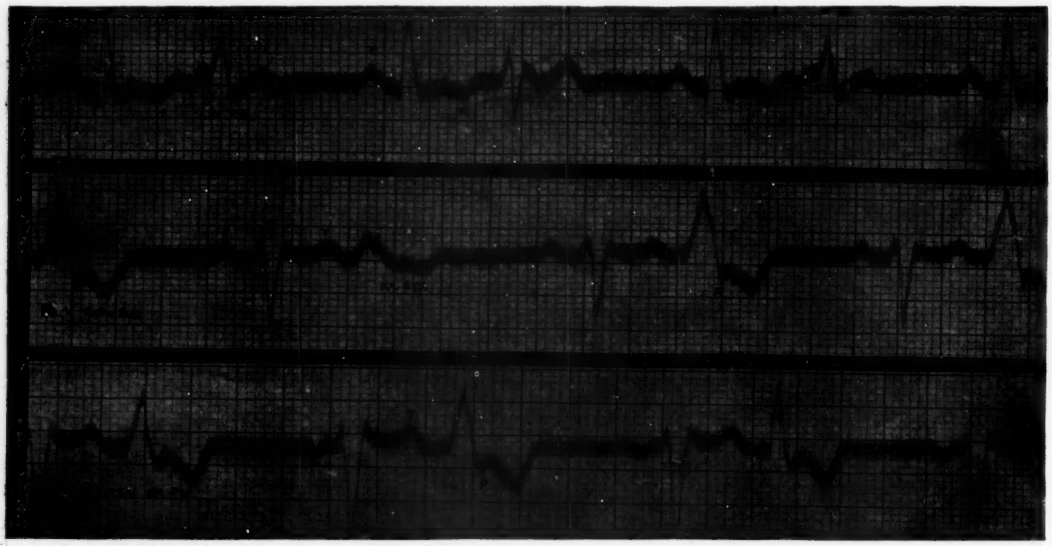

FIGURE IIIB.
Extrasystoles in a male patient, aged sixty-five years, with symptoms of coronary artery occlusion.

accompanied by precordial pain, which may radiate to the left shoulder and down the left arm, causing what Vaquez refers to as the "extrasystolic" type of *angina pectoris*. This condition will cause the patient much misery, and often the medical attendant much worry, because of its close similarity to the pain of true angina or of coronary occlusion. It may be easily distinguished—first the pain is not influenced by exertion, it may arise while the patient is at rest, there are prolonged free periods during which the patient may be quite well, and finally such pain is instantaneous and sudden and coincides with an extrasystole.

In some cases the patient may feel faint during the period subsequent to the premature beat, that is, during the compensatory period. This may be confused with Stokes-Adams disease if the pulse is very slow, on account of the extrasystoles not being palpable.

The treatment of extrasystoles will depend on the underlying cause, such as rheumatic carditis or arterial hypertension. There is no specific treatment for the extrasystole itself, and sometimes it appears to be unaffected by any treatment.

It is wise to eliminate any source of infection which may be supposed to lead to toxic myocarditis, such as bad teeth or septic tonsils. Pay particular attention to the gastro-intestinal tract, see that the food taken is simple, that it is chewed well and that the quantity is not excessive. Drinking with the meals should be forbidden, and smoking and alcohol should be either forbidden or cut down to the very minimum.

Drug Treatment of Extrasystole.

Digitalis is usually not successful in controlling extrasystoles, but it may be given in small doses of 0·3 mil (five minims) three times a day. Quinidine in 0·3 gramme (five grains) doses given twice a day controls some cases. "Luminal" and the bromide of potassium or ammonium will prove of the greatest assistance in those cases with a pronounced nervous element. The main thing is to give the patient reassurance, but many are of such a temperament that they continue to suffer.

A classical example of the benign course which extrasystoles may take, is that of Erb, the famous physician, who observed the presence of this irregularity at the age of twenty-nine; during the next twenty-seven years it in no way impaired his mental or physical vigour. From the age of fifty-six on for the next seven years he was subject to attacks of paroxysmal tachycardia, which were disagreeable, but did not interfere very much with his work or with his sport of mountain climbing. Subsequent to this, there were only occasional extrasystoles. At the age of seventy he underwent a severe gall bladder operation, and died at the age of eighty-three from infective colitis.

Auricular Fibrillation.

I have already referred to the fact of Mackenzie being the first to differentiate auricular fibrillation and to distinguish it from the irregularity of the pulse due to extrasystoles. He recognized it as a condition of a completely irregular pulse; he described its symptoms and ætiology, and first suggested the use of digitalis in its treatment. Auricular fibrillation, before this, was known as *delirium cordis*, and as such had been recognized for many centuries.

Mackenzie at first thought that in auricular fibrillation the auricles were paraylsed, because of the disappearance of the wave from the polygraph tracings. But later, during the course of a *post mortem* examination, he found a dilated and greatly hypertrophied auricle in the heart of a patient who had died of the disease, so he had to relinquish this idea.

Some time later Sir Thomas Lewis operated on a horse with *delirium cordis* and exposed the heart.

The auricles were found to be fibrillating; there was twitching, and convulsive contractions of different fibres were occurring incessantly all over the surface of the auricles, and the auricle made no attempt to contract as a whole.

He called the condition auricular fibrillation, and by means of the electrocardiograph was able to demonstrate that the *P* wave, which corresponds to the contraction of the auricle or to the *A* wave of the polygraph tracing, was absent in this condition, and replaced by small irregular undulations, which he called "*f*" waves; they varied in the different leads, and took place from 450 to 600 per minute. He and his coworkers then showed that in auricular fibrillation and flutter the stimulus to contraction passed down to the ventricle in a completely abnormal manner. He applied the theory of circus movement to these conditions.

In auricular flutter the circulating wave of the circus movement includes the orifices of the superior and inferior *venæ cavæ*, and travels in a fixed path. In fibrillation the path of the circulating wave is shorter and varies with each revolution. The circulating wave gives out impulses at right angles as it passes along, so that the action of the auricles appears to be incoordinated and the muscle fibres appear to contract independently.

This idea of circus movement is not new. In 1876 Romanes found that by stimulating the umbrella of a jelly fish, waves spread out from the site of stimulation, like the ripples when a stone is dropped into a quiet pool. In 1908, Mayer cut a complete ring of tissue from the umbrella and noted that if the ring were stimulated at one point, waves passed round the ring in both directions to meet and extinguish each other at the opposite side.

He then found that by cooling one portion of the ring and applying a stimulus at a point near this, the wave was blocked at the cool area, but passed round in the other direction; if the cooling process was withdrawn so that the cool area became responsive by the time the oncoming wave of contraction reached it, a circus movement could be produced and the wave of contraction would go on round and round for hours. An important factor was found to be the size of the ring—if the ring was too small, the cooled area would not have time to become responsive by the time the wave came round.

Later, Mines, of Cambridge, by rapid electrical stimuli applied in close succession, produced the same type of wave in rings of muscle cut from the hearts of large rays. He considered that the important point was the rapidity of the stimuli, which, occurring at a certain rate, found the muscle on one side refractory and the other side responsive.

In 1887 McWilliam produced fibrillation in the auricles of dogs by faradization. Lewis in turn was able to trace the course of the wave contraction in flutter.

Electrocardiograms of flutter are quite distinctive. In addition to the ventricular complexes, there are small oscillations; they are very constant in the one subject, and though they vary a little in rate, always occur at about 300 per minute. These have been called "flutter peaks".

Auricular fibrillation has also a distinctive electrocardiogram. Small waves again are present instead of the *P* wave, they are smaller and more irregular than the "flutter peaks" and are more rapid, being at the average of 450 per minute. (See Figure IVB.)

Impure flutter is intermediate between fibrillation and flutter, and sometimes occurs in the course of treating auricular fibrillation with quinidine. Quinidine makes these small waves slower and more regular, and they approach in appearance the waves of auricular flutter. This proves that fibrillation and flutter are essentially the same.

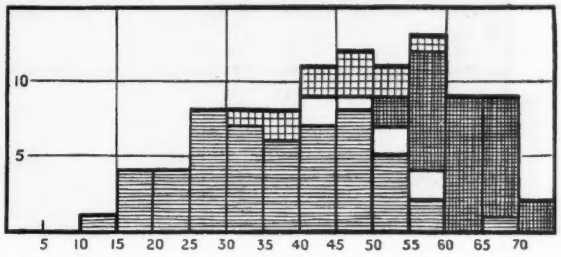

FIGURE IVA.

The ætiology and age incidence of auricular fibrillation (100 cases), shading *et cetera* as in Figure IIIA. (*After* M. Campbell, *Guy's Hospital Reports*, April, 1929.)

The method of spread of the circus wave is interesting. In flutter there is a longish path, extending round the orifice of the superior and inferior *venæ cavæ* and embracing the sino-auricular node. The rate of this wave is such that the refractory period has passed off by the time the next revolution begins; it follows a fixed course, and circulates at the rate of about 300 per minute.

In fibrillation, the course of the wave has not been traced. It is apparently shorter than in flutter, so that the oncoming wave comes to muscle fibres which are almost completely refractory; some fibres will respond, others will not; the course of the circulating wave is irregular and sinuous, so causing the irregular "*f*" waves.

Since auricular fibrillation is probably one of the most common heart conditions which the practitioner is called upon to treat, it is interesting to consider the action of drugs on the circus movement. We must remember that the "gap" is the important part of circus movement. That is, the gap between the oncoming contraction and the refractory period of wave just passed; should this be closed, the movement will stop; if lengthened, the wave will find a shorter route. The variable factors are thus the rate of the circulating wave and the length of the refractory period, both of which can be modified by certain drugs.

Lewis states that the vagus acts differently on the auricular muscle than on the conducting mechanism or bundle of His.

Stimulation of the vagus in fibrillation causes: (i) Shortening of the refractory period so that it lengthens the gap, causing the wave to find a shorter path and thus circulate at a faster rate; (ii) improvement in conduction, so that again the wave tends to circulate more quickly.

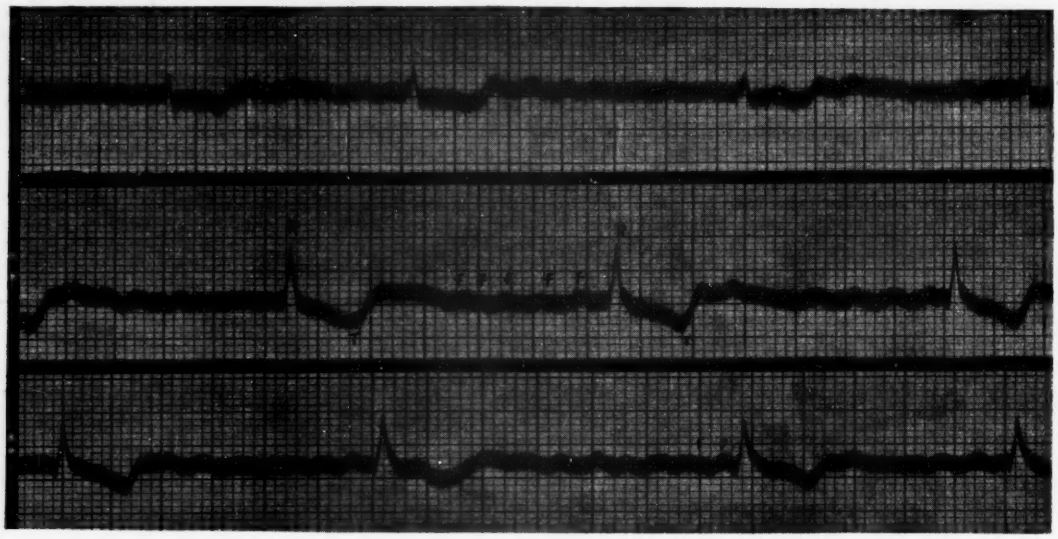

FIGURE IVB.
Auricular fibrillation in a female, aged twenty-six years, with advanced mitral stenosis.

These two actions are antagonistic, so that the result is nil, unless the second effect is greater than the first.

Digitalis stimulates the vagus, quinidine depresses it. Digitalis also has a direct effect upon the auricular muscle; it increases the refractory period, and slows the conduction time, so that its effect is directly opposite to that on the vagus. It will be seen therefore that digitalis will not help to abolish the circus movement in fibrillation; it will only tend to quicken it.

The beneficial action of digitalis in auricular fibrillation is due to its effect upon the bundle of His, by causing a certain degree of blocking, and allowing only a percentage of the stimuli reaching the auriculo-ventricular node to pass on and stimulate the ventricle to contraction. In this way digitalis decreases the ventricular rate, increases the period of diastole, and allows better filling of the ventricular cavities. In auricular flutter, digitalis will tend to increase the rate of the circus movement, and in some cases will produce fibrillation. In many of these normal rhythm is restored when the digitalis is discontinued.

Quinidine is a general heart depressant; it not only depresses the vagus, but it lengthens the refractory period and slows the conduction time; it invariably slows the circus. But for complete cessation the lengthening of the refractory period must be correspondingly greater than the slowing of the circulating wave. According to Lewis, this occurs in 50% of cases. The danger of quinidine in auricular flutter is that the circulating wave may be slowed down to 250 per minute, and induce the ventricle to contract in the ratio of one to one, and so produce ventricular tachycardia, which would quickly lead to failure.

Auricular fibrillation is so extremely common in heart failure that John Parkinson and his coworkers had the greatest difficulty in finding 100 cases of cardiac failure with normal rhythm. Fibrillation has been known to be produced in a healthy heart by an electric shock. The fibrillation of itself must be regarded to a certain degree as a functional disturbance, although it occurs chiefly in diseased hearts. Paroxysmal attacks of fibrillation do occur in hearts which appear to be perfectly normal. The main causes are:

1. Mitral stenosis and rheumatic carditis: probably in all cases of mitral stenosis fibrillation occurs at some period. This also accounts for the great majority of cases amongst young people.

2. Coronary heart disease in older people.

3. Toxic causes: (*a*) Drugs, such as digitalis, which will produce fibrillation in a heart having normal rhythm; (*b*) toxic, in exophthalmic goître; (*c*) acute infections, such as diphtheria, pneumonia, typhoid fever *et cetera*.

Auricular fibrillation is especially prone to occur in mitral stenosis, because of two factors: (i) The presence of the rheumatic virus leading to inflammation of the auricular muscle; (ii) the excessive strain thrown on this muscle by its having to force the blood stream through a narrowed mitral orifice.

In severe cases the rheumatic virus may be so virulent as to cause damage to the conducting mechanism; then we may have auricular fibrillation and heart block together. This distension of the auricle makes it peculiarly susceptible to any increased strain, such as an attack of influenza or over-exertion, when an attack of auricular fibrillation may be precipitated. This irregularity is common in old people and is usually associated with degenerative myocardial changes; it frequently comes on insidiously, and may be associated with a slow pulse, though completely irregular, because the changes in the ventricular walls may embrace the bundle of His and thus lead to some degree of heart block.

Diagnosis of Auricular Fibrillation.

In auricular fibrillation the pulse is completely irregular, the beats varying in rhythm and in volume, and no dominant rhythm can be made out. Its onset is always associated with some degree of congestive failure. It is always advisable to count the heart beats at the apex, as well as to take the pulse, because in these cases the pulse rate may not be a true indication of the ventricular rate. Many of the smaller beats are not strong enough to open the aortic valves and so do not reach the pulse, but they can be felt and heard at the apex. Both the pulse and apex beat rate should be charted when the patient is under treatment; they will be found to approximate as the ventricular rate falls and the beats get stronger.

Exercise increases the irregularity in auricular fibrillation, whereas it obliterates it in sinus arrhythmia and extrasystoles. In about 10% of cases the electrocardiogram is the only means of a definite diagnosis, and can distinguish it from those irregularities due to partial heart block and irregular auricular flutter.

Auricular flutter is probably present when a patient has a pulse rate of 130 to 170, which remains constant in all positions, and continues for over a fortnight. Should he have attacks in which the pulse rate is found to double, the presence of auricular flutter is almost certain.

In flutter we have a comparatively slow regular circus wave which tends to lead to stimulation of the ventricle in a more or less regular way. Most commonly there is two to one rhythm, that is 300 auricular flutters to 150 ventricular contractions. There may be a mixture of two to one and three to one block, leading to an irregular pulse. Sometimes we have four to one, or five to one block, which may lead to a very slow pulse.

Treatment of Auricular Fibrillation.

Mackenzie was the first to suggest the use of digitalis in auricular fibrillation. It is a specific, and frequently leads to spectacular results. Research work of recent years has led to a fairly accurate standardization of the drug. Frogs were at first used for the purpose, now the standardization in America is carried out on cats.

Hatcher and Brody found that it took about 100 milligrammes of dried leaf to kill a cat of one kilogram weight. This amount was called the cat unit. To produce complete digitalization in man it requires about 0·15 cat unit per pound of body weight.

Eggleston has introduced a formula for this calculation. When we remember that 0·06 gramme (one grain) of powdered leaf represents about 0·6 mil (ten minims) of the tincture, it would be thus:

$$\text{Total amount of tincture of digitalis} = \frac{\text{C.U.} \times 0{\cdot}15 \times \text{Wt.}}{100}$$

In a ten-stone man the dose would be

$$\frac{100 \times 0{\cdot}15 \times 140}{100} = 21 \text{ cubic centimetres} = 357 \text{ minims} = \text{nearly six drachms.}$$

At first, Eggleston gave the calculated amount in huge doses, but later modified his treatment in the following way:

(1) *Large Dose Method.* In very severe cases, when one wishes to get the patient under the influence of the drug as soon as possible, one drachm of the tincture is given every six hours during the first twenty-four hours. Then one-half drachm is given every six hours until effect is obtained—this usually takes one to two days.

(2) *Small Dose Method.* When 20 to 40 minims of the tincture are given every six hours, until the effect is obtained, this may take up to ten days.

(3) *The Body Weight Method.* The total amount is calculated, and one-third to one-half of this is given at once. In a ten stone man, two to three drachms; after six hours one-fifth to one-quarter is given, that is, 70 to 90 minims; in six hours again one-eighth to one-sixth is given, 45 to 60 minims, and if more is needed one-tenth of the original dose is continued six-hourly, that is, about one-half drachm.

This method should be employed only in urgent cases, and before proceeding care should be exercised in ascertaining that digitalis has not been given for at least ten days previously. Pardee has found that one minim per pound of body weight is an effective dose, but it must not be exceeded.

Digitalis should not be repeated within six hourly intervals as it takes this time to be absorbed from the stomach.

In extremely urgent cases strophanthin may be given intravenously in doses of 0·6 to 0·3 milligramme (one one-hundredth to one two-hundredth of a grain) or ouabaïn in doses of 0·27 milligramme (one two hundred and fortieth of a grain).

When these larger doses of digitalis are given, the pulse rate and apex beat rate should be taken every two hours.

When the ventricular rate drops to 80 discontinue medication for two doses and once the pulse has been reduced to these proportions, a maintenance dose of 0·6 mil (ten minims) three times daily is usually sufficient.

In patients in whom the tincture disagrees, Nativelle's granules are a reliable substitute and convenient. The large granule of 0·27 milligramme (one two hundred and fortieth of a grain) is equal to fifteen minims of the tincture.

The Use of Quinidine. Quinidine was introduced as a therapeutic agent in auricular fibrillation in the following way.

Professor Wenkeback, in Vienna, was once consulted by a Dutch merchant from the East Indies whom he found to be suffering from auricular fibrillation. The merchant told the professor that he knew he had an irregular pulse, but that he could make it regular if need be. Wenkeback was surprised to hear this, and asked him how he did it, but the merchant refused to tell him, and said that he would return in a few days with a regular pulse. This he did, and after some persuasion he told the professor that he had noticed while taking quinine for an attack of malaria that his pulse became regular. Since then he had taken quinine when his irregular pulse caused him too much discomfort. Quinidine was discovered, in search for a more suitable drug than quinine.

Quinidine has the power of abolishing circus movement and restoring normal rhythm to the heart. It should be used only in selected cases. There were a number of fatalities in the early cases from embolism, because the restoration of normal contraction in the auricles forced out clots from the auricular appendages into the general circulation. Quinidine must not be used in recent congestive failure or in greatly dilated hearts. Both of these conditions predispose to clotting of the stagnant blood in the auricular appendages.

Treat the cardiac failure first with digitalis. When all signs of congestion have disappeared, quinidine may be given.

Always cease the digitalis for at least twenty-four hours prior to commencing with quinidine. Start off with 0·3 gramme (five grains) every six hours, that is, 1·2 grammes (20 grains) in the twenty-four hours. Next day give 0·3 gramme every six hours. On the third day give 0·3 gramme every six hours. On the fourth day give 0·36 gramme (six grains) every six hours. On the fifth day give 0·42 gramme (seven grains) every six hours. On the sixth day give 0·48 gramme (eight grains) every six hours.

Never exceed 2·4 grammes (forty grains) in the twenty-four hours. The pulse will at first rise to 110 or 120, then in favourable circumstances you find one day that the pulse is regular. In many cases the patient is aware of a change having taken place. By this means, you may ward off permanent fibrillation for some years.

It is of special benefit in those patients who have subjective symptoms of the cardiac irregularity. Frequently, a small maintenance dose of 0·3 gramme (five grains) twice a day is necessary.

Some people are susceptible to quinidine, and in some it may produce hyperpyrexia, nausea and vomiting, or skin rashes like measles or scarlet fever.

Paroxysmal Tachycardia.

Paroxysmal tachycardia was first described by Bouveret, and later by Hoffmann, and was ably dealt with by Silberberg, at the Australasian Medical Congress of the British Medical Association of 1929. As the name implies, the condition is one of abrupt sudden onset of rapid heart action, which may continue for a few beats only or which may last for hours or days. The onset is characteristically sudden, the rate remains constant during the paroxysm, and in most cases the offset is again sudden. (See Figure V.)

Paroxysmal tachycardia includes cases of paroxysmal flutter and paroxysmal fibrillation. The great majority of cases consist of runs of extrasystoles. Why a certain part of the heart muscle should thus retain its instability and assume the rate of pacemaker is unknown.

Causes of Paroxysmal Tachycardia.

Paroxysmal tachycardia may be due to emotion as in fear, or the cause may be toxic as in fevers or hyperthyeoidism, or it may be degenerative as in senile hearts or arteriosclerosis. (See Figure VI.)

Some people are greatly affected by the palpitating heart, others appear to be quite unconcerned, many have bad attacks at varying intervals for many years. The chief danger lies in the length of the attack, and the condition of the heart's muscle prior to the attack.

One of Dr. Silberberg's patients, a man of twenty-six, survived an attack lasting for twenty-eight days. (See Figure VII.)

Treatment of Paroxysmal Tachycardia.

We must remember that spontaneous arrest is the rule in paroxysmal tachycardia. Reassure the patient that it is uncommon for death to occur during a spasm. Many of the sufferers learn tricks of their own for controlling the paroxysm, such as bringing up wind, vomiting, tickling the throat with a feather. Vomiting causes vagal inhibition, and a similar result may be brought about by pressure on the vagus in the neck, or by pressure on the eye-balls.

Digitalis in full doses may sometimes stop the attack.

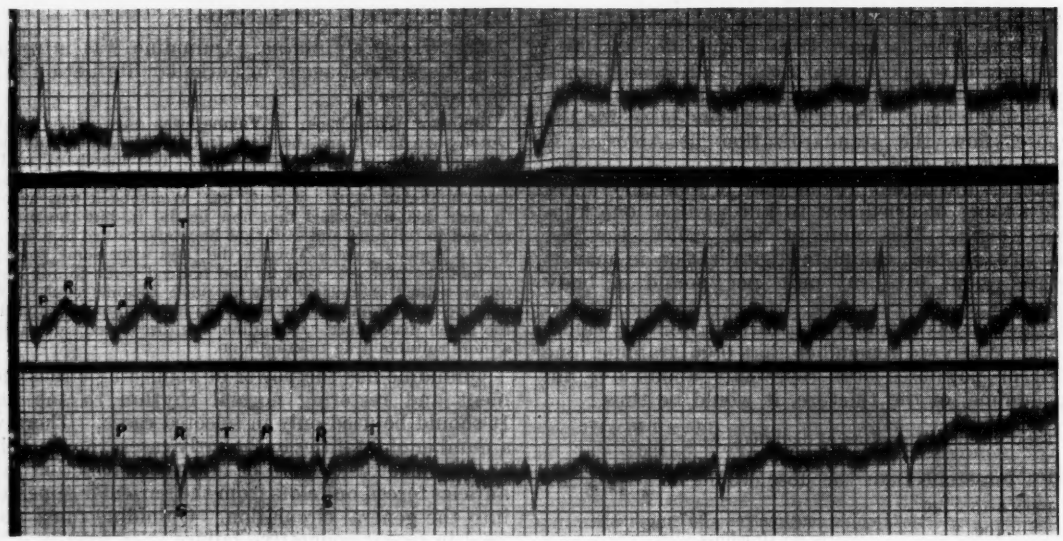

FIGURE V.
Paroxysmal tachycardia in a male, aged eighty-three years. The paroxysm has passed off in Lead III, which shows early heartblock, the $P-R$ interval being prolonged to more than one-fifth of a second.

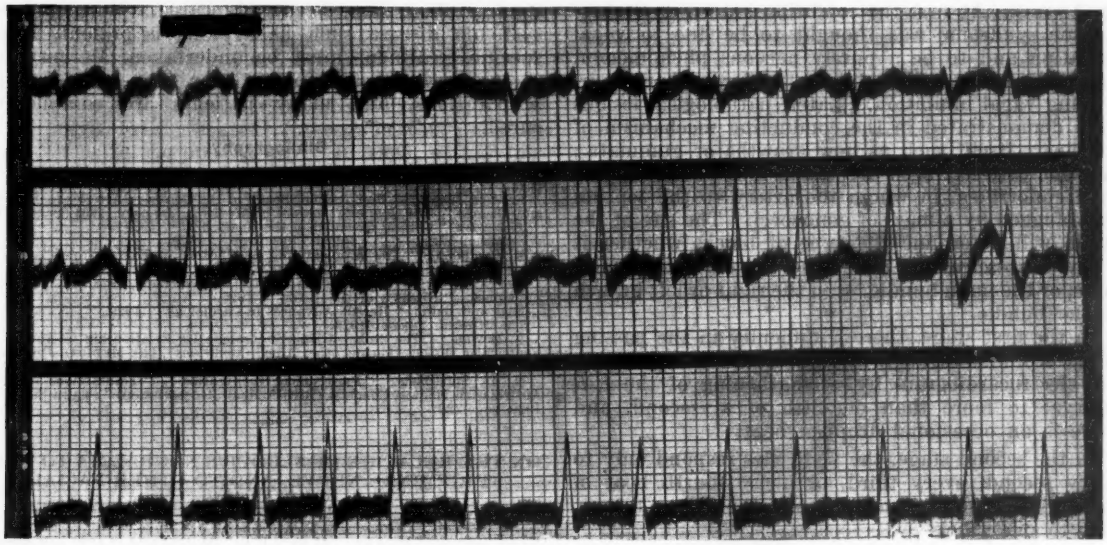

FIGURE VI.
Paroxysmal auricular fibrillation in a male, sixteen years of age, with a recurrent attack of rheumatic carditis and mitral stenosis.

Strophanthin given intravenously in doses of 0·6 to 0·3 milligramme (one one-hundred and fiftieth to one two-hundredth of a grain) is sometimes efficacious, but may be dangerous if much digitalis has been given previously. Quinidine sulphate is the best; inquire for any known idiosyncrasy and if none is present, give 0·18 gramme (three grains) every hour for three to four doses, or 0·3 or 0·36 gramme (five or six grains) every six hours.

Quinidine is dangerous in flutter, and most successful in paroxysmal fibrillation.

"Luminal" or bromide of potash is indicated in the highly nervous patients for relief of distress. In severe cases morphine may be given.

Prognosis depends chiefly on the condition of the heart's muscle before the attack. It is rare for death to occur during the paroxysm, unless definite myocardial degeneration exists beforehand.

Heart Block.

The association of syncopal attacks with a slow pulse, was first described by Stokes, and later again

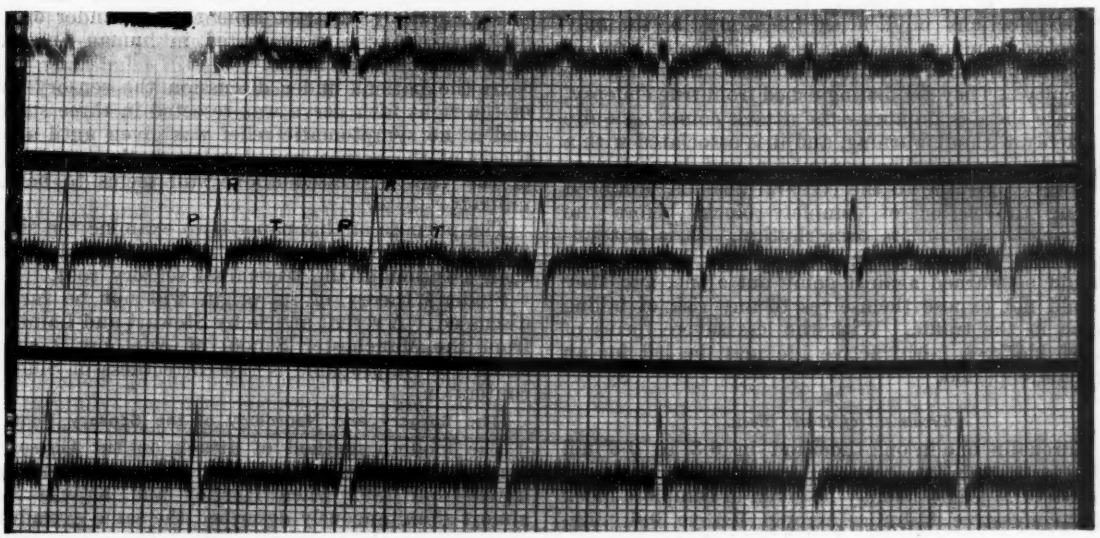

FIGURE VII.
Same case as Figure VI one week after treatment of paroxysmal attack. Note the reappearance of the *P* wave.

by Adams. At first the condition known as Stokes-Adams syndrome was thought to be due to vagal irritation and to be a purely nervous phenomenon. Charcot considered it due to a cerebral tumour, causing vagal inhibition. Later, pathological lesions were demonstrated in the conducting mechanism of the heart itself, such as new growths or gummata; and one case has been described, in a young subject with gonorrhœa, in whom the artery to the septum was plugged by a mass of gonococci. The site of the lesion will determine the type of block, whether it is the sino-auricular node, the auriculo-ventricular node, or the main bundle, or in the right or left branch of the bundle.

In the first stage, there is difficulty in the passing of the stimulus through the *A-V* node, as is shown in the electrocardiogram by a lengthening of the *P-R* interval to more than one-fifth of a second, and there is an occasional dropped beat.

In the second stage, the ventricles respond only to every second, every third, or every fourth beat of the auricle, thus producing two to one, three to one, four to one block.

In the third stage, there is complete ventricular dissociation, and the auricles and ventricles beat on at their own rate—the auricles at seventy-two per minute and the ventricles at twenty-five to thirty-five per minute. The electrocardiogram has helped to reveal bundle branch block.

Causes of Heart Block.

The causes of heart block are as follows: (i) Acute infections, such as rheumatism, diphtheria, pneumonia *et cetera*. (ii) Chronic infections, such as syphilis. (iii) Degenerative changes — coronary sclerosis or fibrous myocarditis. (iv) New growths or hydatid cysts—situated in the bundle of His. (v) Digitalis in large doses.

The important features in heart block are: (i) The condition of the heart muscle generally; in acute infections mild heart block may be the only sign of some myocardial damage. (ii) The occurrence of syncopal or epileptiform attacks may lead to difficulty. They are distinguished from true epilepsy, because there is no aura—the attack comes on quite suddenly without warning, and it is associated with a very slow pulse. The convulsion and loss of consciousness are due to the cerebral anæmia produced during the period of complete block, before the ventricle takes on its own rhythm. It is not uncommonly fatal. Patients who suffer in this way should be warned against going out alone, and they should not expose themselves to any risk of falling down in the traffic. With the less severe forms of heart block, a fairly active life may be led for an indefinite period.

Treatment of Heart Block.

Adrenalin one in one thousand in 0·3 to 0·6 mil (five to ten minims) doses in the attack is frequently successful. Digitalis may be given in complete block and congestive failure. Atropin 0·4 to 0·6 milligramme (one one-hundred and fiftieth to one one-hundredth of a grain) will act in those cases associated with vagal irritation. Barium chloride 0·12 gramme (two grains) has been advocated. When some systemic disease such as syphilis is present, antispecific treatment must be undertaken. Mercury in these cases appears to be safer than the arsenic preparations, and many patients are completely cured.

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ON THE RELATION OF SLUDER'S NEURALGIA TO THE TRIGEMINAL NERVE, AND TO OTHER FACIAL NEURALGIAS.

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IN a recent issue of this journal[16] a case of Sluder's neuralgia following an infection of one sphenoidal sinus was described and discussed. As the condition was relieved by alcohol injection of the fifth nerve in the region of the *foramen ovale*, and not relieved by Sluder's technique of injection of the spheno-palatine ganglion, it was held that this condition is in reality a subtype of ordinary fifth nerve neuralgia due to an infective neuritis of certain fibres in the nasal sinuses. It was suggested that, as the pain was mainly centred about the eyeball, the particular fibres affected may be a derivative of the archiac deep ophthalmic nerve, which runs between the olfactory and optic capsules. This nerve is phylogenetically distinct from the remainder of the fifth nerve and is represented in human anatomy by the naso-ciliary nerve.[1]

In the patient under discussion the sensory root of the right fifth nerve was resected in three stages, and as no symptomatic relief was given until anæsthesia of the cornea had developed, the correctness of the view expressed above still seems to be not unlikely.

The purpose of this paper is to examine the situation of the fibres affected in Sluder's neuralgia and their position in the trigeminal sensory root.

Sluder's theory of its origin will not be further discussed. A recent article by Stewart and Lambert, from the Manchester School of Anatomy[2] shows again how improbable is the correctness of Sluder's contention.

The Surgery and Neurology of the Sensory Root.

The modern method of attack on the Gasserian ganglion for relief of ordinary trigeminal neuralgia dates from 1892 when Hartley and Krause each almost simultaneously described the same extradural operation by the temporal route. This, in its initial description, merely consisted of resection of the individual branches of the fifth nerve, but Krause

in 1893 showed that the ganglion itself could be removed. Sir Victor Horsley first suggested section of the posterior root, and operated on one patient who died. In 1901 Spiller and Frazier reported the first successful intracranial division of the sensory root, and Frazier's great interest in this operation continues to the present day. In 1925, with Whitehead,[3] from morphological studies he concluded that the three peripheral divisions were fairly accurately localizable in the posterior root and could be separately resected. His method of partial resection still remains the popular method of attack.

Dandy,[4] however, in 1929 described the result of his method of resection of the sensory root at the pons by an occipital approach and his latest method of partial resection suggests that the pain fibres are carried in the posterior part of this root but have lost, by now, the peripheral arrangement that Frazier has shown.

The Gasserian ganglion consists of a semilunar mass of cells, from the concavity of which the sensory root emerges. In section these cells show as a narrow crescent, upon the convex surface of which the three sensory divisions converge. The motor root is a separate bundle passing beneath the ganglion from the mesial to the lateral surface, where it ultimately unites with the mandibular division and supplies the muscles of mastication. As it passes under the ganglion, it receives fibres from the middle and maxillary part of the ganglion. The sensory root, as it passes to the pons, is a somewhat rounded strand of fibres, which after piercing the dura becomes gradually flattened and finally enters the pons as a vertical ribbon in a sheath of arachnoid. A variable number of accessory fibres are placed somewhat more ventrally in relation to the motor root.[5]

Frazier and Whitehead, in 1925, from morphological studies, showed that the ophthalmic or mesial part of the ganglion develops somewhat separately from the other portions. They concluded that the sensory root still fairly accurately presented the peripheral arrangement and that the outer strands represented the mandibular division, the mesial the ophthalmic, while the maxillary division lay between. Frazier, in his operation of partial resection of the root, claims to be able to resect more or less whichever part of the peripheral fibres he desires, and so to leave a minimum of anæsthesia.

His diagrams from a recent article[6] are appended and are self-explanatory. (Diagrams I, II and III.) They show clearly a distribution of anæsthesia somewhat similar to the classical and well-known distribution of the individual peripheral nerves.

Dandy, however, is disinclined to believe that the individual peripheral supply can be so schematically represented in the posterior root. His doubt lies in carefully controlled neurological observations of anæsthesia on a number of patients the posterior roots of whom he has wholly or partially resected at the entry into the pons. In his early cases he resected the entire sensory root as he saw it. A few patients had total anæsthesia over the whole of the fifth area, but many to his surprise showed retention of touch and temperature sense in various combinations and positions. The only consistent loss was in the perception of pain. An anatomical study of the area showed various aggregates of accessory fibres running from the sensory root, anterior to the main root, and undoubtedly the reason for this dissociation

DIAGRAM I.
Area of anæsthesia five days after section of the outer third of the sensory root. (*After* Frazier.)

of anæsthesia is the passage of varying proportions of fibres through these accessory roots.

He was then led to resect only the posterior part of the main root. By this method he found that he

DIAGRAM II.
Area of anæsthesia ten days after section of the middle fasciculi of the sensory root. (*After* Frazier.)

abolished pain only and would leave ordinary epicritic sensations unaffected. Hence, the patient may find loss of his neuralgia with practically normal sensation of his face.

Dandy finds no definite evidence of peripheral zones in his partial resection, but notes that the bulk of sensory loss seems to take place in the first or upper division. He criticizes Frazier's view as a result of these operations, but admits that some rearrangement of fibres may have occurred by the time the pons is reached.

If we accept Frazier's views as at least approximately correct, they do not, on thought, in any way clash with Dandy's findings. The operation of resection of the posterior root is performed close to the ganglion. The sensory root, after it reaches the pons, divides into an ascending and a descending root. The descending root, which is long and connects with the upper spinal segments, is largely protopathic and subserves the sensation of pain and the

DIAGRAM III.

Area of anæsthesia five days after section of the ophthalmic portion of the sensory root. (*After* Frazier.)

temperature sense. The ascending root is epicritic and serves the sensation of touch. It is phylogenetically more recent and has developed along with the discriminative cortex, a point which has been clearly shown by Brouwer. Between these two fixed points there must be a rearrangement of fibres into those which are mainly epicritic and pass more directly to the thalamus and the cortex, and those which possess more feeling tone and pass by the longer route of the descending trigeminal tract.

Phylogenetically, the fifth nerve is a fusion of many sensory roots corresponding to the various cranial segments.[7] In cyclostomes and fishes there is a separate nerve, the deep ophthalmic nerve, which runs between the olfactory and optic capsules. It represents the naso-ciliary nerve in part in man and has been suggested as forming with the third nerve the sensory and motor components of the preoral area. The fifth nerve, on the other hand, is the nerve of the oral region and a nerve of two parts, one anterior to the oral cleft forming the maxillary nerve, the other branch posterior, forming the mandibular nerve. The supratrochlear, frontal and supraorbital branches of this first division are phylogenetically branches of the above divisions, while the naso-ciliary nerve corresponds to the old deep ophthalmic nerve. Dixon's study of the development of the branches of the fifth nerve, based on serial sections of His's embryos, seems to make this point clear.[8]

Symptomatology.

Sluder's neuralgia[9] follows no definite peripheral nerve distribution. Following an infection, usually of the sphenoidal sinus or posterior ethmoidal cells, a neuralgia remains which centres about the eyeball and radiates concentrically therefrom, so that frontal, temporal and maxillary areas may be encroached upon. If the neuralgia is severe, it may radiate to the mandibular division or to the opposite side of the face.

In addition there is a fairly characteristic occipital pain, which may radiate, as the severity increases, to the neck, the shoulder and even the arm and hand.

The patient's history was previously described; and in this instance, following a presumably blood-borne infection of the right sphenoidal sinus, a severe and persistent Sluder's neuralgia followed.

After a series of fruitless attempts at injection according to Sluder's plan and technique, the fifth nerve at the *foramen ovale* was successfully injected under local anæsthesia by Dr. Julian Smith. The first drop of alcohol violently increased the pain, then came partial anæsthesia and relief. After six months the pain returned and was similarly treated. At the time of publication of the previous article the pain had not returned after a spell of eighteen months, but shortly afterwards recurred and soon became almost unbearable.

The hyperæsthesia, which had been previously clearly mapped out, had increased in area concentrically, with radiation to the left side of the face.

Dr. Julian Smith was called upon to face the problem of relief once more, but it was found quite impossible, owing to the dense fibrous tissue about the *foramen ovale*, to infiltrate the ganglion successfully, although three attempts were made.

Realising that the particular fibres affected were in all probability in the mesial edge of the root, total resection was decided upon, as any risk was little to the patient as compared with the intolerable pain from which she was suffering.

On May 14, 1931, Dr. Smith approached the ganglion by the usual temporal route using ether anæsthesia. The bone was abnormally thick and the ganglion seemed unusually adherent, which may possibly have been a result of the previous infiltration with alcohol. Hæmorrhage was troublesome and after considerable difficulty on this account, a large part of the lateral portion of the root was resected. Unfortunately, for the same reason, no further strands could be seen, and the operation was concluded with a mutual feeling of hope that the resection might have extended as far as the mesial strands. The patient, on the first glimmer of consciousness, was interrogated as to whether her neuralgia had ceased, and immediately answered, "No".

The sum total of result next day was found to be anæsthesia of the first and the lower portion of the second division (Diagram IV) and the pain was as constant and as severe as ever, with the exception that radiation did not extend to the anæsthetic areas. Six days later the wound was reopened and several large strands were resected further mesial to those already divided. Upon regaining consciousness the patient said that pain was still present and was rather worse. Anæsthesia now reached to the lower lid and by comparing this diagram with Frazier's, it will be seen that the anæsthesia just corresponds to the area of resection of the outer two-thirds of the root, or resection of those fibres corresponding to the second and third divisions. (Diagram V.)

On May 25, a third operation was attempted and one small strand, about half the size of a match, was found still further mesially. The patient was then lightened in anæsthesia until the left conjunctival reflex was plainly obtained while the right reflex was found to be absent. This was regarded as a probable indication that the whole root had been resected. Upon the patient's regaining consciousness the neuralgia had entirely

disappeared, and with it, the occipital and cervical radiation. (Diagram VI.)

Upon a careful neurological examination, however, it was found that the upper area of the first division was normal and the extent of the new anæsthesia was an area involving the lower

DIAGRAM IV.
Area of anæsthesia to cotton wool and pin prick after section of outer strands of the sensory root with no relief of the neuralgia.

lid, the conjunctival surface of the sclera of the eye, but with some preservation of sensation in the cornea itself. In addition the upper lid was slightly hypoæsthetic. The sphenoidal sinus was found to be entirely anæsthetic, as was the main portion of the nasal mucosa, except in its uppermost portions.

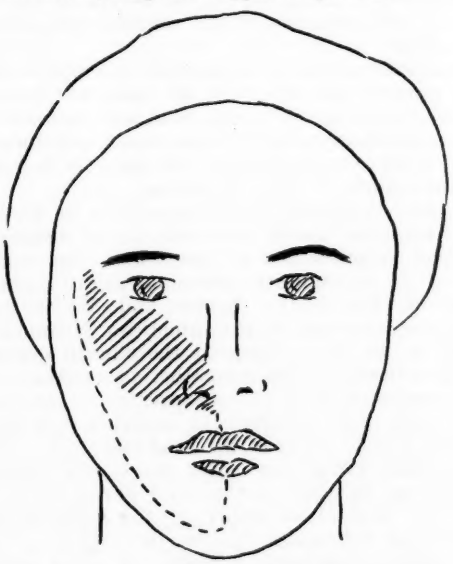

DIAGRAM V.
Further anæsthesia produced by section of further strands, with no relief of the neuralgia.

This result, occurring more or less fortuitously, seemed to indicate clearly that the neuralgia was associated with nerve fibres lying between the first and second divisions of the fifth nerve. The mucosa of the sphenoidal sinus is credited with innervation from both first and second divisions. The naso-ciliary branch of the first division supplies the sphenoidal sinus and the posterior ethmoidal cells by its posterior ethmoidal branch, while the second division, by small orbital branches passing by way of the spheno-palatine ganglion, similarly supplies the mucosa of those areas. Whether these particular branches are in relation within the posterior root cannot be shown. The naso-ciliary nerve certainly supplies the eyeball. The orbital branches of the second division are not known to do so. When one considers the symptomatology of this condition in this particular instance, with pain in the eyeball, radiating therefrom, subsequent to a solitary empyema of one sphenoidal sinus, a neuralgia of the naso-ciliary division does not seem unlikely.

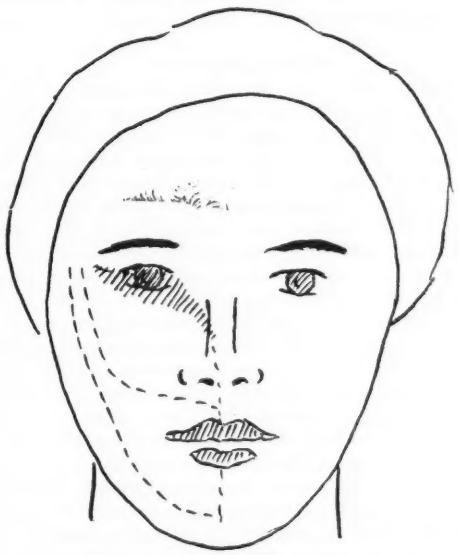

DIAGRAM VI.
Further anæsthesia after section of a strand placed further mesially, with relief of the neuralgia.

As mentioned in a previous article, any results that Sluder obtained by his method of injection may have occurred through interference with those branches passing by way of the spheno-palatine region. If these fibres are developmentally part of the naso-ciliary complex, their irritation might produce a similar neuralgia.

As was previously shown, the occipital pain is a pure radiation. Interference with the nucleus of the pain in the fifth nerve area immediately prevents it. However, many cases of sphenoidal sinus infection have been described in which the pain seems to be largely occipital or occipito-vertical, and it does not seem unlikely that a neuralgia confined to this area might not be produced from this source. The explanation of this lies in the most caudal part of the descending fifth nerve nucleus, which lies in the first and second cervical segments, the area of nerve supply of the occiput. This part of the spinal nucleus has been shown to be in the main associated with the first division of the fifth nerve. Hence, parts

of the fibres subserving pain sense in this area are relayed in the upper cervical area. Again, part of the sphenoidal bone is associated with the occipital bones, in developing from the notochordal region. The occipital bones are essentially structures of cervical nerve innervation. It may be that pure occipital pain of sphenoidal sinus origin represents a somewhat "post-fixed" innervation of this area. The relation of the first division of the fifth nerve to the cervical nerves is not only well shown anatomically by the anastomosis of the supraorbital branches with the occipital nerves, and by the course of the long recurrent meningeal nerve to the tentorium, but also clinically by the reference of frontal and supraorbital headache to the occiput and by the radiation of the pain of occipital neuralgia to the brow area. Recently a typical "tic" of the first division of the fifth nerve was found to possess trigger areas in the occipital zone.

Atypical Neuralgias.

Unfortunately all so-called neuralgias of the eyeball or surrounding parts, even if they radiate to the occiput and present the typical syndrome, are not examples of Sluder's neuralgia any more than all the neuralgias of the lower trigeminal area are *tic douloureux*. Application of the former term must be definitely limited to neuralgias of the typical distribution, following infections of the nasal cavities. The very common form of neuralgia or headache occurring in migrainous subjects may very closely resemble Sluder's neuralgia, including the radiation to the occiput. Such a condition usually occurs in attacks and may be accompanied by the other manifestations of migraine. At times, however, a type of neuralgia occurs which may persist for days or weeks and which is located in the region of the eyeball, with or without occipital pain. It is particularly apt to occur in overwrought women of middle age, and is extremely intractable of treatment. In the presence of positive skiagraphic findings, it is hard to resist the temptation to operate on the paranasal sinuses, usually without result. In the majority of cases the findings are normal. Such cases are usually classified as neuralgia of migrainous type. Interference with the Gasserian ganglion is not usually considered, mainly because of the nervously exhausted type of patient, the presence of remissions, particularly with betterment of the mode of living, and the fact that often the neuralgia is not confined to one eye, but is apt to occur in the other, although predominantly occurring in the one.

Possibly some of these neuralgias may be infective conditions of the naso-ciliary nerve or its branches. The theoretical possibilities for such an infection would not only involve its terminals in the nasal mucosa but in the eye itself.

The typical migrainous cases are certainly not of an infective type, and their origin remains, like that of the origin of all migraines, obscure. Theoretically a spasm of part of the arterial supply of the orbit, or the engorgement subsequent to such a spasm, might be considered as a cause.

Resection of the root of the fifth nerve does not abolish entirely deep orbital sensation any more than it does deep facial sensation, and pain may be noted on movement of the eyeball even following a complete section. Harris[10] notes that such an operation or injection abolishes only certain headaches of a non-migrainous type. Parker, from the Mayo Clinic,[11] in a survey of certain atypical facial neuralgias, reissued the warning that this particular operation is specific only for *tic douloureux*, a fact which has been corroborated often since.

Atypical neuralgias in the fifth nerve area, whether migrainous or otherwise, are not only usually unrelieved by any operation producing anæsthesia of fifth nerve type, but are frequently aggravated. If any such operation is considered, it is advisable to study the effect produced by a well placed local anæsthetic either in the region of the *foramen ovale* or more peripherally before surgery is attempted.

One reason at least for this is that the sensory supply of the facial area is greater than has been considered. Since the operation of resection of the posterior root of the trigeminal nerve has become popularized in the treatment of major trigeminal neuralgia, it has become almost universally noted that in this section certain fibres associated with deep pressure sense and deep pain persist. Lesions which in addition interrupt the facial nerve, usually produce a complete anæsthesia. It is, therefore, held that the facial nerve carries these particular sensory fibres. Pain, again, may be noted at times occurring with movements of the eyeball after complete fifth nerve post-Gasserian division. It is obvious, therefore, that there are other sensory fibres within the orbit than those of trigeminal origin, but exactly within which nerve trunk these lie must be still somewhat speculative. The theoretical possibilities are the facial nerve, the nerves of the eye muscles, and branches of the cervical sympathetic to the orbit.

The author knows of no clinical observation that would support the view that the facial nerve enters into the sensory supply of the orbit, but anatomically the temporo-facial branch has many anastomoses with fifth nerve terminals, and it is unlikely that such junctions should be entirely motor.

The rôle of the cervical sympathetic in atypical facial neuralgias has of late come under discussion, with the recrudescence of interest in the sensory fibres of the involuntary nervous system in general throughout the body. Mixter and White[12] at the annual meeting of the American Neurological Association in 1930, discussed the clinical evidence for pain pathways in the sympathetic nervous system. They concluded that pain-bearing fibres probably run in many of the sympathetic branches that course along the blood vessels of the head and neck. They believe these fibres may enter the central nervous system from the first to the third dorsal segments, and their method of study is the paravertebral injection of "Procaine" into the region of the appropriate sympathetic ganglia. If relief occurs, they inject alcohol, resect the ganglia, or sever the *rami communicantes*. They report two cases of facial neuralgia in one of which pain was still relieved three months after operation, but in the other only part of the occipital pain was relieved, with persistence in the face. They also report relief in four cases of typical facial neuralgia. In the subsequent dis-

cussion the question was raised as to whether such results actually indicated interruption of sensory fibres, or merely some alteration in the vascular supply of the part. Grant reported relief of facial pain for a period of twelve months following thoracic ganglionectomy in one case, but a recurrence after three months' relief in another.

The various reports of port-arterial sympathectomy for atypical facial neuralgias do not seem to be very encouraging, and although it seems that interference with the sympathetic outflow may in some way affect facial pain in certain cases, the reason is not entirely clear. Again, before surgical operation is attempted, it would be advisable to try the method of trial and effect with local anæsthesia, remembering even then that the effects may not be permanent after operation.

It has been frequently noted that two types of fibres occur in the nerves supplying the ocular muscles. In addition to the ordinary large medullated fibres, many small fibres, medullated or non-medullated, may be seen. Boecke's conception of these non-medullated fibres as being sympathetic in origin has been challenged, and the majority of observers, including Sherrington, consider them to be sensory. Wilkinson,[13] in an excellent study using the Bielschowsky method of staining in serial section, has investigated them in the superior oblique muscle in cats, and considers that they are proprioceptive in origin. In the main these fibres pass to the brain stem by way of the fourth nerve, although occasionally a few fibres are seen to be derived from the ophthalmic division of the fifth nerve. This small branch has been noted as well in man. Usually these fibres are seen to leave the nerve again, but occasionally they could be traced to epilemmal proprioceptive nerve endings. He did not note ganglion cells along the course of the nerve, but a series of oval cells between its entrance into the brain and its nucleus.

Woollard[14] has recently succeeded in keeping alive a cat for thirteen days after resection of the third nerve close to the brain stem. By the toluidine blue method, after alcohol fixation, he found chromatolysis in the main third nerve nucleus, with the exception of the posterior group of cells, and incidentally in the adjacent mesencephalic root of the fifth nerve, a tract which is now known to contain proprioceptive fibres from the muscles of mastication. He concludes from this and other experiments that the fine fibres are sensory in origin, and possibly are derived from the mesencephalic root of the fifth nerve.

If Woollard's work be confirmed, it would mean that the great sensory column associated with the trigeminal nerve may act as a relay station for other sensory fibres than those of fifth nerve origin, and the explanation of some atypical facial neuralgias would be more apparent.

The relation of the most caudal part of this column to the upper cervical segments has been commented upon. Nageotte[15] clearly demonstrated its association with the so-called sensory or gustatory nucleus of the seventh cranial nerve, and it now seems possible that within it may yet be found associations with sensory fibres derived from the various motor ocular nerves. This would be well in accord with Gaskell's view[7] of it as a fusion of the sensory roots of numerous cranial segments. Individual variation would naturally occur as elsewhere in the body. The sense of taste amply demonstrates this, where three separate paths are used in varying proportions, that is, the ninth, the seventh and the fifth nerve. Here the sensory column described by Nageotte in detail is clearly demonstrated to be associated with all three nerves, although mainly facial in origin.

The Psychic Factor.

The question of the psychical origin or continuance of atypical facial pains is extraordinarily difficult, as elsewhere in the body. It has been frequently demonstrated that section of numerous posterior roots, or even hemisection of the cord, has been insufficient to relieve some persistent somatic pains. The same fact cannot be denied to the facial region.

Some of these patients are neurotics from the inception. Many appear to be of normal nervous calibre, although they may eventually become grossly neurotic under the burden of the constant facial pain and repeated operation, with the disappointment of failure. Such a group is the type of facial neuralgia which may follow antral operation. In two of these patients seen, one derived no benefit from alcohol injection of the Gasserian ganglion with excellent anæsthesia, while the other was quite unaffected by total section of the posterior root.

It would seem in some of these patients as if every possible afferent sensation from a part is harnessed to the area of the brain concerned with the perception of pain to that part, and that, even in the absence of the part itself, sufficient afferent channels remain from elsewhere to keep alive the sensation.

The whole question of pain of psychic origin is very confused. While one may surmise that a constant summation of painful stimuli may produce microscopical changes in connected cell groups of the central nervous system, there undoubtedly exists a type of individual of high intelligence, who, if he has sufficient insight, and a sense of humour, will admit that he can manufacture pains in any part at will. If an organic pain of some duration occurs in that individual, it may be extremely difficult to eradicate its effects, even if the lesion itself be apparently cured.

Summary.

1. Section of the posterior root of a patient with a Sluder's neuralgia following an empyema of one sphenoidal sinus is described. The operation was done in three stages, but no effect was produced until the conjunctiva became hypoæsthetic.

2. It is suggested that the condition is a neuralgia of the naso-ciliary nerve of infective origin.

3. Atypical facial neuralgias, their relation to Sluder's neuralgia, and to the known anatomy of the facial region are briefly discussed.

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JOHN ABERNETHY, 1764–1831.[1]

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IT seems appropriate that some recognition of the founder of British clinical surgery should be given by us this evening, since this year marks the centenary of his death. In these days of relatively intensive education of the medical profession as a whole, as well as of the lay public, the problems encountered and overcome by the stalwarts of our surgical world of one hundred years ago are difficult to visualize. Modern hospitals with their wealth of equipment, orderly routine, skilled nursing, special departments and large staffs of well-trained specialists representative of all sections of the medical profession, cause little if any comment; both we and the general public take it for granted that all that now is, should be. Therefore, it is a good thing to inquire occasionally into the conditions pertaining in times not so very far distant and on this occasion I ask you to go back with me to some consideration of the surgical world when John Abernethy at the age of 23 years was elected an Assistant Surgeon to Saint Bartholomew's Hospital, in 1787.

A surgeon conducting an operation in those days had to recognize that there was no security against the simplest and most successful procedure being followed by dangerous and often fatal results. When he had closed the wound he was, as Volkman remarked, "like a husbandman who, having sown his field, waits with resignation for what the harvest may bring, and reaps it, fully conscious of his own impotence against the elemental powers which may pour down on him rain, hurricane or hailstorm".

There were four chief "hospital diseases" which constituted the nightmare of operating surgeons, namely, erysipelas (or St. Anthony's fire), septicæmia, pyæmia and hospital gangrene. No theory had been brought forward which afforded any rational explanation of their nature, and surgeons submitted to them as inevitable. The only undisputed fact was that their onset was favoured by dirt and overcrowding, especially among patients who had suppurating wounds. It was assumed consequently that pollution of the atmosphere was the chief thing to be avoided.

[1] Read at a meeting of the Section of Medical History and Literature of the New South Wales Branch of the British Medical Association on November 25, 1931.

John Abernethy was one of the most clear-sighted men in his time, as were John Hunter and Lister in theirs, and he saw that air alone was not the prime factor. The particular observations that influenced Abernethy in this belief pertained to the condition of surgical emphysema and to that of pneumothorax, as in each case no inflammation occurred as a result of the presence of air in abnormal situations so long as there was no associated external wound whether accidental or made deliberately in order to let the air escape. However, argument from these somewhat rare cases did not appeal to many, and Abernethy along with his contemporary surgeons remained in the dark as to the exact causation of these inflictions.

The medical profession of those days was comparatively ignorant of anatomy and still more ignorant of physiology; its members were only reluctantly shedding the ancient traditions and shibboleths of the bygone ages. Surgery had no scientific basis before the days of John Hunter. He collected facts and collated data and left the broad foundation upon which his pupils raised that building which subsequent advances have brought to its present state—the edifice of modern surgery.

John Abernethy was one of John Hunter's most attentive and admiring students, but, as he himself admits, he did not then recognize Hunter's genius, although he regarded him as a paragon and one upon which to mould his own career. A chance remark of Hunter's that his "mind was like a beehive" impressed Abernethy very much and he thought the simile admirable, "for in the midst of buzz and apparent confusion there was great order, regularity and abundant store of food which had been collected by incessant industry".

Such, in brief, were the conditions existing when Abernethy, at the age of fifteen, was apprenticed to Sir Charles Blicke, of Saint Bartholomew's Hospital, and first became interested in the teachings of John Hunter, who was to prove the dominant factor in moulding his career. Abernethy was the son of a London merchant and grandson of that John Abernethy who rent the Irish Presbyterian Church into sections of subscribers and non-subscribers by remaining as a minister at Antrim when the Synod had ordered him to Dublin. He is said to have derived from his grandfather not only a peculiar obstinacy, but also his love for the medical profession. During his apprenticeship he attended lectures by Percival Potts, he learnt his anatomy at London Hospital, where he was prosector to Sir William Blizzard, and John Hunter taught him pathology and showed him how morbid and comparative anatomy might help to advance surgery. It is interesting to note that Abernethy was born just one year after John Hunter settled in London and his opening contribution to science, "Surgical and Physiological Essays" in 1793 was coincident with the close of the labours of his illustrious friend.

Abernethy was Hunter's immediate successor and apparently constituted himself a sort of champion of his master's physiological theories which he dramatized in the lecture-room by his poetic imagination and vigorous style of delivery. He soon attracted students to his lectures given at his home in Bar-

tholomew Close and when he was elected to the staff of Bartholomew's Hospital in 1787 his pupils followed him; their number increased to such an extent that it was found necessary to build a special lecture theatre four years later. One of Abernethy's contemporaries states that his most striking characteristic was "a memory alike marvellously ready, capacious and retentive—qualities common enough separately, but rare in powerful combination". As a lecturer he had a rare command of language and expression, and was able to hold the interest of his audience and send them away with a good conceit of themselves. He was one of those independent thinkers who have the power to override the most established conventionalisms with opinions of his own which he was able to support in no uncertain fashion.

Sir Benjamin Brodie remarks:

He kept our attention so that it never flagged and what he told us could never be forgotten. He did not tell us so much as some other lecturers, but what he did he told us well. His lectures were full of original thought, of luminous and almost poetical illustration, the tedious detail of descriptive anatomy being occasionally relieved by appropriate and amusing anecdotes. Like most of his pupils I was led to look up to him as a being of a superior order and I could conceive of nothing better than to follow in his footsteps.

Abernethy had very lofty views upon the nature and duties of the medical profession and in these days of commercialism it does one good to read certain of his opinions and beliefs.[1] Abernethy was meticulous as to his duty to the hospital and to his patients there. However much he might forget the courtesy due to his private patients, he was never unkind or brusque to those whom charity had confided to his care. One morning when leaving his home to attend the hospital a message came that someone wished to see him forthwith, to which he replied: "Private patients, if they do not like to wait for me can go elsewhere, but the poor devils in the hospital I am bound to take care of."

Although appointed as Assistant Surgeon to Saint Bartholomew's at the age of 23 years, it was not until 28 years later that he attained the status of full surgeon, being then 51 years of age. Although this period of hospital service afforded him a certain field of observation and did not altogether preclude him from putting his principles to the trial of experience, it did much to restrict his activities in the actual practice of surgery and so narrowed the scope for his inquiries that he was forced to carry out many of his experiments in his own home.

The rapid advance in chemistry at this time had attracted Abernethy, and he was interested in its application to the composition of animal tissues. He devoted a considerable amount of time and labour to his chemical experiments, as may be gathered from one inquiry as to the effects upon animals of vegetables grown in air and distilled water as compared to the normal. For this purpose he fed rabbits exclusively on cabbages grown on flannel moistened with distilled water, and considering the appetite of rabbits and the rate of growth of cabbages raised in this way he must have been kept very busy. He was very interested also in the function of the lymphatics, or as they were then called "the absorbents", and by dissection and injection experiments on the whale, the horse and other large animals, he proved that the lymph glands were not made up of convoluted absorbents, but were of a cellular structure. It was about at this time also that his attention was directed to improvement in the method of treating lumbar or psoas abscesses, which were regarded then as a very serious malady, for if the abscess burst or was opened, severe constitutional symptoms developed and as a rule ushered in "the slow minings of the hectic fire" which ultimately destroyed the patient. He was emphatic as to the constitutional origin of the malady and insisted on the need for greater attention to the general health of the patient, but his treatment by aspiration before signs of inflammation appeared proved a real advance.

As another example of his tenacity of purpose we may consider one of his experiments dealing with the function of the skin in which, as a result of holding his hand under a glass jar filled with mercury, he collected in sixteen hours a half-ounce measure of gas. As a result of an analysis of the gas collected, he concluded that "the air perspired consists of carbonic gas a little more than two-thirds, and of nitrogenous gas a little less than one-third". In the same patient manner he carried out other experiments on the power of the skin to absorb various gases, and found that eight ounces of oxygen were absorbed in eight hours, three ounces of nitrous gas were absorbed in five hours, one and a half ounces of hydrogen were absorbed in five hours, and that one ounce of nitrogen was absorbed in eight hours.

Coincidently with these experiments on the skin Abernethy conducted others with regard to the excretory powers of the lungs, and thus began the reasoning he developed very fully at a later date of the interdependence of various organs and their relationship to the bodily functions as a whole. This led on to the elaboration of an hypothesis which he never seemed tired of enunciating, namely, that an organ which is apparently the seat of disease may not be the seat of the original cause of that disease, but really a secondarily affected organ; so that treatment of the obvious lesion may lead one to believe a disease is incurable because the real cause has never been attacked.

Abernethy has been erroneously criticized as having ascribed all ailments to disorder of the digestive organs, but from the experiments alluded to one can see that his view was a much broader one than this, although in actual practice disorders of the alimentary tract were so common that undue advertisement was thereby given to his theories. In fact, one historian definitely states that "Abernethy believed that all local diseases were either of constitutional origin or due to digestive disturbances and in practice treated nearly everything with calomel and 'blue mass'."

At the age of 30 years Abernethy had definitely established himself as a coming man of no ordinary talent both on account of his lectures and of his writings. At the hospital consultations his *confrères* attached value to his opinion and his influence on the development of the hospital was considerable. He had a masterly way of analysing a case and presenting the important points in such a manner that from doubt and confusion of ideas he shaped an easy,

definite and intelligible proposition. One of his very practical achievements in operative surgery was not the ligature of the external iliac artery for aneurysm, but the very clear and explicit directions he laid down for trephining the skull in cases of head injuries. All patients with head injury up to this time had been subjected to trephining, but Abernethy pointed out that disappointment was the rule unless there were definite indications of pressure on the brain either from a depressed fracture or intracranial hæmorrhage and that mere lifting of the bone was not the essential factor.

In 1800, at the age of 36 years, Abernethy married Anne Threlfell, whom he had met at a patient's house where she was entrusted with part of the nursing. He was much gratified by her kindness and attention to the patient and when added to these qualities which he always admired, there were "personal attributes of no mean order and lively, lady-like and agreeable manners" Abernethy became much attracted by her. However, he was a shy and sensitive person in social matters and being wholly absorbed in teaching, studying and practising his profession, he chafed at the usual method of time-consuming courtship, and to cut the Gordian knot of his difficulties proposed marriage by letter, apologising for so doing and asking her to take a fortnight for consideration. The lady must have understood him well, for she replied in the affirmative and apparently raised no objection to his dispensing with a honeymoon and continuing his usual hospital lectures even on the day of his marriage.

All through his life he was most attentive to his work at the hospital, both as regards the care and treatment of his patients and the preparation and delivery of his lectures. Although frequently brusque in manner and impetuous, his emotions were easily stirred by pain and distress in his patients. As he neared middle age he evinced greater irritability and discussed this with his friends on several occasions. In his early life he had expressed disgust at the manner in which he had seen patients caressed and humbugged and flattered by pompous and indifferent medical practitioners and he had made up his mind to avoid such cajolements at all costs. This possibly accounted to some extent for his manner, but he does not seem to have cared much for private practice, and once observed: "I tried to learn my profession and, thinking I could teach it, I educated myself to do so; but as for private practice I am obliged to do that too." As for operations *per se*, Abernethy held that they occupied such a low place in the duties of a surgeon and had so little to do with the science of the medical profession that there was little in most of them to off-set that repulsion which was inspired both by his science and his humanitarian instincts. As he advanced in years this repugnance to operation increased and he was apt to be fidgety, impatient and unpleasant to his assistants, although he was never unkind to the patient—in fact it was usually an additional trial to him when a patient bore pain with fortitude.

One day he was to perform a severe operation on a woman, and before commencing spoke a few words of encouragement to her according to his usual custom. She stood the pain stoically, but after a short time remarked: "I hope, sir, it will not be long", to which Abernethy made earnest reply: "No, indeed, that would be too horrible".

In his dealings with patients he tried to make things clear to them and as he became busy and had no time for lengthy explanation he referred them to his book, "Abernethy's Surgical Observations". He did this to try to abolish the absurd idea that there is something in medical practice inappreciable by the public, and he also maintained that "if a medical man thought he had done his duty when he had written a prescription and the patient regarded his as fulfilled when he had swallowed it, they were both deceived". At all times he tried to stimulate the patient to an understanding of his malady and called for intelligent help in the treatment.

One wonders what he would think of the ultra-specialization that exists in the medical profession today, when considering his remark that "the division into medicine and surgery is mischievous as directing the attention of the two orders of practitioners too exclusively to the diseases usually allotted to them". This opinion, however, was modified by him later on and in the Hunterian Oration which he delivered in 1819, he gave a definite approval of this division of interests, but deprecated further subdivision.

In 1814 Abernethy was appointed Professor of Anatomy and Surgery at the College of Surgeons, and on account of the necessity for regular lectures there, he added a further burden to an already strenuous life as by then he had an enormous following at his hospital lectures and the previous year had accepted the surgeoncy of Christ's Hospital, which he held until 1828.

He stands really as the founder of the Medical School of Saint Bartholomew's Hospital and, as has already been stated, his views on treatment were based more on physiology and pathology than upon the results of operation. As a scientific surgeon he followed John Hunter, though he had neither the originality, application nor intense industry of his master. He became the foremost exponent of Hunter's views and it was he who paved the way for the Hunterian traditions in surgery to develop right up to the present day.

Strange as it now seems to us, Abernethy loomed larger in the eyes of his contemporaries than did Hunter—to his students because of his wonderful gifts as a lecturer, to his colleagues because of his studious life, his sterling honesty and his great independence of character, and to the general public for his bluntness of speech and eccentricity of manner which made him a topic of general conversation.

It seems tragic that Abernethy should have become the sufferer from a complaint which we now believe would have been prevented or partly cured by the very principles he himself enunciated. For many years he was troubled with what seemed to be chronic infective tonsillitis with occasional peritonsillar abscess and as the years went on he became a martyr to chronic rheumatism which was probably influenced to some extent by the full-bodied port wine on which he prided himself. In 1824, at the age of 60 years, he resigned from the hospital since he held

that all appointments should terminate at that age to give younger men an opportunity. However, he was prevailed upon to continue his work, which he did for another year or so; finally he relinquished duty early in 1827. His infirmities grew apace and in 1829 he resigned from his professorship at the College of Surgeons and retired from practice. He lingered on in suffering, which he bore with a resignation unexpected in one of his character, and died tranquilly in the spring of 1831.

Striking testimony as to his influence at Saint Bartholomew's is given by Sir James Paget, who joined the staff in 1834:

> The hospital had then begun to decline among the schools chiefly due to constant dissension and mischievous rivalry among the teachers. Since Mr. Abernethy's retirement there had been no one willing to do the constant routine work of management in the school and who was at the same time either strong enough or pliant enough to get his own way. Thus it was that the school declined in members and in fitness for teaching, though it still offered more opportunities than enough for those who could teach themselves. No one was in any sense responsible for the students and the helps to learning were very defective except as regards the museum to which Abernethy had donated his private collection which he had used at lectures.

In conclusion, I would quote further remarks of Paget regarding Abernethy:

> He should be remembered not as rough wit or a mere eccentric, but as a man of rare sagacity, an admirable surgeon and teacher and one of the most graceful writers of his time.

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THE FABLE OF THE LOADED CAECUM.

By HUGH C. TRUMBLE,
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IT appears to be a fairly widely accepted view that in man in the upright position the colon tends to sink in the abdomen, that when it does so it drags on various structures, giving rise at times to all sorts of troubles, and that "loading" of the colon increases the drag and aggravates the symptoms. At first sight this seems to be reasonable enough, but a little reflection will show that here again things are not what they seem.

The colon is but one of a number of structures enclosed in the abdominal cavity. There is room in the lower part of this space for some only of the viscera. Other things being equal, the most dense, not necessarily the largest, structures will eventually settle to the bottom, whilst the less dense will float, as it were, upon them. Many of the solid organs, such as the kidneys and liver, are handicapped by more or less firm fixation to the abdominal walls and are thus out of the competition. Of the more mobile structures it is of interest to consider and compare the intestines, large and small.

The specific gravity of the gut wall, whether removed at *post mortem* examination from cadavers or from freshly killed animals (rabbits and guinea-pigs), was about 1·06. The liquid content of the small gut, freed from gas, varied from just over 1·0 to 1·02. The specific gravity of the contents of the proximal colon similarly freed from gas varied between 0·97 and 1·05, usually being nearer 1·0 than 1·05. When, however, masses of formed or pultaceous fæcal material were thrown into water, they almost invariably floated. This was largely due to the presence of gas in the interior of the fæces. Segments of human gut removed after ligation at each end almost invariably floated in water, and in some cases the specific gravity of the ascending colon and caecum with contents was as low as 0·5. In order to avoid the possibility of error from *post mortem* production of gas, observations were made in two rabbits and two guinea-pigs freshly killed. The small gut, removed *in toto*, sank in water, but some small segments of ileum, which could be seen to contain gas, floated when separated from the main mass. Gas was seldom present in the upper part of the small intestine. The distal part of the colon containing formed fæces invariably floated strongly in water and sometimes in paraffin of a specific gravity of 0·86. The proximal part of the colon floated in water in all cases but one (rabbit), but sank in paraffin.

In passing, it is of interest to note that the specific gravity of the kidney is practically identical with that of the gut wall, whilst liver tissue is somewhat denser (about 1·09).[1]

Thus it would seem that segments of the gut are most dense when empty, less dense when containing liquid material, still less dense when only formed fæces are present, and least dense when there is an admixture of free gas. Now, the small intestine, especially the ileum, usually contains liquid material and not much gas. The right-sided colon always contains liquid material and usually a large amount of gas. In the passage of the transverse colon the contents tend to become more and more inspissated, so that in the descending colon fluid is not normally found, the fæces being semi-solid and formed. Gas is again present in greater or less quantity, either free or imprisoned in the fæcal material.

In the upright position coils of small intestines, which are mobile and relatively dense, will tend to fill the true pelvis and pile up in the central part of the abdomen, to the exclusion of less mobile and less dense structures, including the colon. Further, when there is much gas present in the colon, the latter may even be buoyed up so as to exert an upward thrust upon the viscera above it. Free gas will not be distributed throughout the length of the colon. In the upright position it will naturally make for the flexures, and here it may be demonstrated usually by the fluoroscopic screen. Sometimes gas in the region of the splenic flexure produces the appearance known to radiologists as cascade stomach. The central part of this organ is buoyed up by the distended colon and when an opaque meal is swallowed it collects in the proximal pouch so formed until a certain level is reached, when it spills over the colonic impression into the distal segment. In the recumbent position the gas should tend to pass from the splenic

[1] These observations were taken at room temperature (about 65° Fahrenheit scale). At body temperature the expansion of contained gas would tend to reduce the specific gravity of a viscus still more. No attempt was made to allow for the difference between the pressure at which the observations were made (that is, atmospheric pressure) and that in the interior of the gut in the living animal.

flexure into the descending and transverse segments, whilst fluid, if present and unimpeded, should take its place. When opaque liquid and gas are introduced this may be seen actually to happen. Normally, however, at the splenic flexure, the contents of the gut are not liquid and so probably do not move in the gut in response to change of position, but gas probably does move. The contents of the right-sided colon are always liquid and gaseous. Thus the density of this section is relatively low compared with that of segments of small gut. In the upright position liquid will tend to run to the caecum and possibly to the middle of the transverse colon, whilst gas will pass to the hepatic flexure. Its buoyant effect must tend to float the right-sided colon upwards upon the relatively dense coils of small gut. It is worthy of note that the terminal segment of the ileum passes up from the bottom of the pelvis to meet the colon.

It should be noted that barium sulphate, which is the material usually administered by mouth to provide a radio-opaque meal, is a very heavy substance, its specific gravity being 4·48. The specific gravity of the suspension in use at the Alfred Hospital is 1·26. This is much in excess of the specific gravity of any of the abdominal viscera. It is possible that water is absorbed in the passage of the meal along the gut, thus rendering the specific gravity of the artificial content even higher. In any case, the presence of barium sulphate in a coil of intestine must increase its density to a degree never approached under normal conditions. A caecum filled with this heavy material may attain a position in the abdomen much below that which it would ever reach at any other time, by displacing coils of gut which are for the moment relatively less dense. It is fallacious to conclude that a portion of gut normally occupies the position in which it is seen to be when full of barium sulphate in suspension.

It is almost inconceivable that the colon should ever displace the small gut from the lower part of the abdomen in the upright position. It is relatively less dense, and also less mobile. On the other hand, it is possible that under certain conditions the colon may fall in relation to fixed structures such as the spine, the great vessels and nerves of the visceral plexuses and, hence, that it may drag on these and other structures. In the first place the small intestine may at times be more or less empty, so that its volume is small and insufficient to fill up the lower part of the abdomen and provide support for the colon. Normally, of course, the space vacated by the small gut should be "taken up" by the contraction of the abdominal wall musculature, so that the colon would be supported as before. Secondly, when the abdominal wall is lax and sags abnormally, the lower abdominal space is greatly increased in volume, so that the small gut does not fill it and room is left into which the colon may sink.

If the above reasoning is sound, it follows that the colon cannot sink low enough to drag on its attachments unless there is a general subsidence of the intestines into an abnormally large lower abdominal space. Treatment should then be directed towards the reduction of this space, and will include measures designed to increase the power of the spinal, abdominal and thoracic muscles, such as exercises and massage; and passive support by some form of belt. However it be effected, diminution of the volume of the lower part of the abdomen will result in raising of the colon and abolition of such symptoms as are due to the drag it exerts upon its moorings. If the colon is filled with radio-opaque material and examined by screening in the upright position, it may be seen to rise in the abdomen when pressure is applied to the abdominal wall just above the *symphysis pubis*. All mobile parts of the colon, as well as the flexures, are affected. If symptoms are not abolished, they are not due to the low position of the colon, and operative measures aiming at high fixation of the gut are doomed to failure. Indeed, there seems to be little justification for any form of colopexy under any circumstances.

Apart from the above considerations, it is held by an ever-increasing number of observers that the symptoms usually grouped together and described under the headings of visceroptosis, *caecum mobile* and so on, are not due to the low position of the colon in the abdomen. This seems to be no more a factor in the production of symptoms of intestinal stasis than are the bands and kinks of Lane, and operations designed to correct the former will probably go the same way as those which formerly were performed to eliminate the latter. Those readers who are interested will find the whole question very fully and critically discussed by Bedingfield in his recent book, which is well worth reading.[1]

It is interesting to note that both in the erect and recumbent positions there are influences at work tending to keep the flexures at a relatively high level. In the erect position gas tends to collect at the flexures, thus buoying them up. In the recumbent position the flexures are lower or more posterior than the caecum, transverse colon and descending colon, so that gas tends to leave and fluid to gravitate to them. Whether these factors have had anything to do with the shape of the large gut and the position of the flexures is a moot point.

Reference.

[1] H. Bedingfield: "Visceroptosis and Allied Abdominal Conditions Associated with Chronic Invalidism", *Oxford Medical Publications*, 1930.

Reports of Cases.

ACUTE VESICULAR DERMATITIS CAUSED BY CURAÇAO ALOES.[1]

By IAN HAMILTON, F.R.C.S. (England),
Adelaide.

A MARRIED woman, aged forty-nine, had suffered from varicose eczema of both legs for the last fifteen years. When I visited her with a view to treating her varicose veins, she gave a history that four days previously she had been given some "capsules of pure herbs" by a so-called violet ray expert. She took two of these capsules, with an interval of twelve hours between them, and within twenty-four hours was covered by a rash.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 26, 1931.

The patient's eyelids were swollen, and the face, arms, legs, groins, grooves under the breasts and the front of the chest were thickly covered by minute vesicles such as are seen in herpes. Over the front of the chest and over the dorsa of the feet these vesicles had become confluent, the surface epithelium lost and an acute eczema resulted. The worst part was where the nutrition of the skin had suffered on account of the varicose veins. Recovery was slow, but uninterrupted.

Mr. W. T. Rowe, Government Analyst, very kindly identified the substance as powdered curaçao aloes. The official dose of aloes is 0·12 to 0·3 gramme (two to five grains); the larger capsules contained 1·2 grammes (twenty grains), so that in a single dose the woman received four times the maximum dose, repeated in twelve hours.

Mann and Brend[1] do not mention aloes as a poison and neither Hale-White[2] nor Martindale[3] mentions any specific ill effects from an overdose of aloes.

References.

[1] Dixon Mann and W. A. Brend: "Forensic Medicine and Toxicology", 1922.
[2] W. Hale-White: "Materia Medica", 1927.
[3] Martindale and Westcott: "The Extra Pharmacopœia", 1924, Volume 1.

TORSION OF THE UTERINE ADNEXA.

By CECIL COGHLAN, M.B., Ch.M. (Sydney), F.R.C.S. (England), F.R.C.P.I., D.G.O. (Dublin), F.R.A.C.S.,

Honorary Assistant Gynæcologist, Saint Vincent's Hospital; Honorary Gynæcologist, New South Wales Community Hospital, Sydney.

TORSION of the uterine adnexa associated with a pathological condition of the ovary or tube, although not rare, is relatively uncommon, but torsion of the normal tube and ovary in virgins with no demonstrable preexisting pathological condition is extremely rare. It was my fortune to have one of each of these types of adnexal torsion on the same day at Saint Vincent's Hospital in February last.

Case I.

The first case is one of torsion of the right Fallopian tube and ovary associated with bilateral ovarian cystadenomata.

Miss E.L., aged twenty-nine, was admitted to Saint Vincent's Hospital on February 20, 1931. She gave a history of severe right-sided pain for two days, about one year previously. Two weeks before admission she had a sudden, very severe pain which doubled her up. The pain continued severe for two days and she had frequent vomiting. The pain then eased a little, but was still fairly severe and was worse on walking. The menses were regular every twenty-eight days, lasted for seven days, were moderate in amount, but were accompanied by some pain in the lower part of the abdomen. The last menstrual period occurred on February 7, 1931. She had had no other illnesses. On examination of the abdomen there were tenderness and hypertonicity in the lower right quadrant. A vaginal examination disclosed the uterus pushed forward against the pubes and, posterior to it, a very tender double cystic swelling.

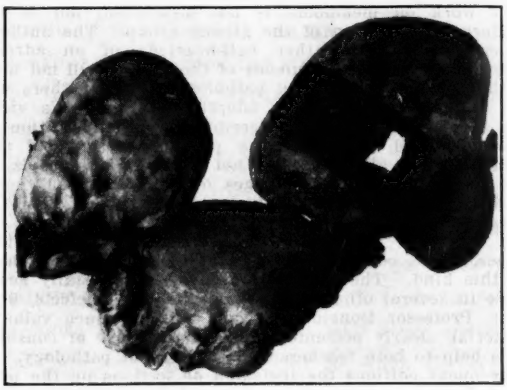

FIGURE I.
Specimen removed in Case I—twisted ovary and tube on the right.

On February 26, 1931, under "Avertin" anæsthesia, subtotal hysterectomy with double salpingo-oöphorectomy was performed. The right tube and ovary were twisted two and a half complete turns in a clockwise direction.

Pathological report on the left ovary was that a large retention cyst was present with several smaller ones and a cyst of the *corpus luteum*. The right ovary was large and cystic, dark in colour and contained blood clot. No evidence of malignant disease was found.

Case II.

The second case is one of primary torsion of normal tube and ovary in a virgin.

Miss L.B., aged nineteen, was admitted to Saint Vincent's Hospital on February 26, 1931. Five days before admission she had a very severe pain in the left iliac region which continued with more or less severity for four days and then became more severe, doubling her up, so that she could hardly walk. The day before admission the pain passed to the middle and then the right side of the abdomen, growing more and more severe. She was constipated, but there was no vomiting or nausea. Menstrual history was normal. The last menstrual period occurred on February 14, 1931.

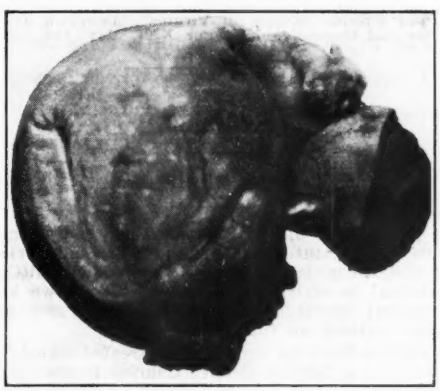

FIGURE II.
Specimen in Case II.

On examination there was great tenderness over the lower part of the abdomen, with rigidity and hyperæsthesia, especially on the right side. The hymen was virginal. The temperature was 37·8° C. (100° F.), the pulse rate was 98. Operation disclosed a fair amount of blood-stained exudate and a large (four-inch) blue-black cystic mass in the right lower abdominal quadrant, which proved to be the left tube greatly dilated and twisted one and a half turns counter clockwise. The left ovary was black, twice its normal size and included in the volvulus. The right tube and ovary appeared perfectly normal and there was no evidence of pelvic infection. The appendix was normal. The left tube and ovary were removed.

The pathological report was that the specimen consisted of a Fallopian tube, greatly distended in its distal portion and with the fimbriæ glued together. The distal portion was blue-black in colour. Attached to the specimen was an ovary, also enlarged, and of a blue-black colour.

Sections showed massive organized and unorganized blood clot in both tube and ovary. Villi in the proximal part of the tube appeared normal. No evidence of inflammatory or decidual reaction was present.

Discussion.

Torsion of the normal Fallopian tube in virgins is very rare. Darner,[1] in 1926, could find only six authentic cases in the literature, and Downer and Briner,[2] in 1931, confining their study to patients under seventeen years of age, could find only sixteen cases, ten of which involved both the ovary and the tube.

In considering the ætiology of this condition one must remember that the tube is normally a very mobile structure, lies usually in a position posterior to its attachment to the uterus and inclined medially. There is therefore a twist of up to 90° in a normal tube. Various theories are advanced as to why this normal twist should be increased, but none are very satisfactory. They are well discussed in Darner's article.[1] The twist is almost invariably clockwise on the right side, and counter clockwise on the left. The symptoms frequently simulate those of acute appendicitis. I am reporting these cases owing to the extraordinary coincidence of having both on the one day at the one operating session, and on account of the rarity of the condition occurring in the second case.

Acknowledgements.

My thanks are due to the staff of the Pathological Department of Saint Vincent's Hospital for their careful examination of the specimens, and to Dr. E. M. Humphery for photographing them.

References.

[1] H. Lauran Darner: "Torsion of the Normal Fallopian Tube", *American Journal of Obstetrics and Gynecology*, Volume XI, March, 1926.

[2] M. G. Downer and Osborne A. Briner: "Torsion of the Undiseased Uterine Adnexa in Virgins", *American Journal of Obstetrics and Gynecology*, Volume XXI, May, 1931, page 665.

Reviews.

STAMMERING.

THE joint effort of medical man and educationist has given us "The Nature and Treatment of Stammering", by Boome and Richardson.[1] It is a well-balanced little book, non-technical in style, and is the best book we know to give medical practitioners a sympathetic and also an instructed outlook on the problem.

The authors have for several years treated school children and adults at a London County Council Remedial Centre. Patients have had the good fortune to be treated along the lines of the newer psycho-pathology, and excellent results have been obtained. The extension of such coordinated work between medical men and educational authorities would prove indeed a blessing to sufferers from this affliction, but the difficulty is to find workers qualified for the task.

The authors correctly point out that the subject has for the most part been ignored by general practitioner and specialist alike, and that the treatment of stammerers has for many years been in the hands of quacks and charlatans. "The instruction given and treatment prescribed . . . actually confirms and aggravates the difficulty . . . . It is the universal custom to teach stammering under the guise of curing it." Stammering is classed among "speech defects", like lisping and defective articulation, whereas it should be under some such heading as "nervous disorders".

Causes of stammering may be divided into the endogenous or constitutional, and the exogenous or environmental. Stammerers are, except for the affliction, the same as other people. The chief trouble is fear, or dread of speaking, and an inferiority complex is developed. A feature of the book is a chapter written by the patients themselves, which presents an illuminating insight into the real nature of the trouble—the distressing state of their mental world.

Treatment by the authors has correctly proceeded on the principle of taking the mind of the stammerer off his affliction. He must cultivate a "saturated feeling of mental ease" and let the utterance of his thoughts look after itself.

Treatment proceeds along the lines of relaxation, suggestion and rhythmic coordination. Each stammerer's peculiar history is elicited, his psychic errors are brought into the open and removed, and his environmental conditions are harmonized.

The authors do not refer to one factor of success—the tremendous psychic relief experienced by the absence of concealment of the stammer, the absence of reserve and the new and helpful atmosphere. The gradual building up of new confidence and the development of conscious power are other psychic effects which can be obtained by a carefully framed range of speech work.

There does not appear to be sufficient appreciation of the stammerer's very real difficulties when he gets outside the remedial school. The book is, however, only brief in content, and is a most commendable illustration of happy results obtained from the developing new technique of the medico-psychologist.

[1] "The Nature and Treatment of Stammering", by E. J. Boome, M.B., Ch.B., D.P.H., T.D., and M. A. Richardson; 1931. London: Methuen and Company, Limited. Crown 8vo., pp. 143. Price: 3s. 6d. net.

MORBID ANATOMY AND THE MICROSCOPE.

PROFESSOR DONALDSON'S "Practical Morbid Histology" is, as its name implies, a book to be used in conjunction with a microscope.[1] It is written concisely and clearly, and most of the illustrations are accurate and informative. The ground covered includes most of the commoner inflammatory, neoplastic and degenerative lesions, but there are some important omissions. Thus, while much space is devoted to the relatively unusual bone dystrophies, osteomyelitis and bone tuberculosis and all diseases of joints are entirely ignored. Other important subjects not dealt with are hyperplasia and chronic cystic disease of the breast, traumatic fat necrosis, alimentary polyposis, carcinoid tumours of the intestine, and many of the general features of the metastasis of neoplasms. Masson's important work on melanoma is not mentioned, nor is the modern classification of the glioma group. The author's acceptance (albeit rather half-hearted) of an adrenal origin for the Grawitz tumour of the kidney will not meet with the approval of most pathologists, while others will not endorse the too ready adoption of Sampson's views on endometriosis. The description and illustration of chordoma fail to convey the peculiar features of this tumour, which also is perhaps not the "very rare neoplasm" the author asserts. The pages on "endothelioma" serve once more to denote the confusion of the subject. The two lengthy final sections, one on histological technique and the other on parasitic worms, together occupying fifty pages, might be discarded without serious loss to a book of this kind. The information involved is readily accessible in several other works. Despite these defects, however, Professor Donaldson's book contains much valuable material, clearly presented, and should prove of considerable help to both teachers and students of pathology. In subsequent editions the inclusion of sections on the principal bone and joint diseases would greatly enhance the usefulness of the volume in this respect.

[1] "Practical Morbid Histology: A Handbook for the Use of Students and Practitioners", by R. Donaldson, M.A., M.D., Ch.B. (Ed.), F.R.C.S.E., D.P.H., with foreword by Sir Humphry Rolleston; Second Edition; 1931. London: William Heinemann (Medical Books) Limited. Crown 4to., pp. 497, with 214 illustrations. Price: 42s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 27, 1932.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE SEMINAR.

ALL medical practitioners know that if they would keep up with the river of progress that leads eventually to the ocean of greater knowledge, they must keep as far as possible to the centre of the stream. This stream arose from very small beginnings in the mountain tops of the long ago; as it passed onwards there came places where the water looked as if it were still, but the flow went on. Sometimes rivulets joined it to swell its volume—the rain of intuition fell and the snow of constant endeavour melted—and the stream increased. There were times when the stream flowed quickly; there were times when rocks in its course made as though to stop it, when its face was no longer peaceful, when the rapids of discord threw it foaming and angry onwards to become, however, the same peaceful stream as before. The river of progress will continue to flow; its tributaries have been many and more await it; and those who steer a course in mid-stream will be the first to reach the ocean. In every part of its course this stream has been bordered by reeds and grasses, where water, almost stagnant, caught in eddy and backwash, has lain. To be careless in steering a course is to run the risk of being left in the backwash.

The medical practitioner who wishes to keep abreast of progress in medicine, is faced by a difficult task. The task is made easier if he has adequate library facilities. The position of the Australian medical practitioner is different from that of his brother in Great Britain. In the Commonwealth of Australia medical library facilities are woefully inadequate, and medical practitioners who wish to examine the literature on a special subject, find themselves severely handicapped. This defect has been discussed in these pages on several occasions and suggestions have been made which might bring about an improvement. Even if medical libraries were well stocked with all the important journals and with the latest books, they would not be of much use to medical practitioners in their systematic reading. Medical libraries are intended primarily for reference; if they were to be used for systematic reading, they would have to be lending libraries and would be almost useless for reference purposes. Medical practitioners in city or suburb who read an abstract of an article in a journal to which they subscribe, can refer to the original paper if the journal containing it happens to be among those filed at the British Medical Association Branch or University Library. Country practitioners have not this advantage, and it is with the object of stimulating the country practitioner that this article is written.

Most medical practitioners open their medical journals to read articles on subjects in which they are interested. Those who do not open their journals need not be considered at the moment, for they will not read these words. But reading, to be effective, must be systematic. The rush and hurry of modern life, the commercial element which has crept into medical practice and which threatens to reduce it to the level of a trade, social distractions such as the bridge table, which become a curse when carried to excess, these and a hundred and one other factors all combine to lure the busy practitioner from systematic study. Those who have had to prepare a paper for a medical gathering, know what a stimulus is provided by a definite objective. This stimulus may be had wherever "two or more are gathered together" by the institution of a seminar. Medical practitioners who agree to form a seminar

or study circle meet at regular intervals. Each member of the group undertakes to present to the other members the salient facts gathered from his reading in one of the branches of medicine or in a particular journal. This recital is followed by an extempore discussion. Each member of the group should agree to subscribe to a different medical journal and the journals should be available to every member. In this way overlapping of journals is prevented and the members enlarge the scope of their reading at a small cost. If medical practitioners, particularly in the country, institute study circles, they will avoid the backwash and the still waters among the reeds and grasses; they will also find a *camaraderie* that will do away with jealousies, misunderstandings and bickerings.

Current Comment.

PRIMARY CANCER OF THE LIVER.

In the Croonian Lectures delivered before the Royal College of Physicians in June of last year and referred to in a recent issue, Matthew J. Stewart dealt with precancerous lesions of the alimentary tract.[1] He stated that the most hopeful aspect of cancer research is that connected with the demonstration of local causative factors and the prevention of precancerous lesions. This, of course, applies chiefly to the more superficial kinds of malignant disease, which, however, constitute only a small proportion of the whole. Precancerous lesions of the alimentary tract do not afford the same opportunities for prophylaxis. Stewart made special reference to cancer of the liver, primary carcinoma of that organ being rare in white-skinned races. In Leeds, from 1910 to 1931, 14 cases were discovered at 12,800 autopsies. If seven doubtful cases were added, the percentage was 0·16. Excluding persons under thirty years of age, the percentage becomes 0·25. The incidence among some coloured races is greater: in Chinese and Javanese it is 2·3% and among South African natives 2%. The Hungarian percentage is 0·3. Among South African natives J. H. Harvey Pirie found 36 primary cancers of the liver in 91 carcinomata. In the Dutch East Indies, of 447 tumours examined, 83 were primary cancers of the liver, almost the whole of which were from Chinese and Javanese patients, amongst whom they represented 57% and 32% of all tumours examined.

Different explanations have been given of this racial incidence. In South Africa and the Far East hepatic cirrhosis associated with schistosomiasis is held to be a possible factor. In China another fluke, *Clonorchis sinensis*, may be responsible. E. P. Snijders and M. Straub suggest that repeated and chronic affections of the sources of the portal vein often present in the native races of tropical countries may play a part in the causation of hepatic cirrhosis and cancer. Van Dort suggests that the different incidence in different countries of primary heyatic cancer may have a racial basis. In Europe the incidence is higher in Magyar and Mongolian races than in Aryan stock. There is a high rate in Madras (peopled by Dravidians), but a low rate in Calcutta and Colombo (inhabited by Aryans). The rate amongst negroes, Chinese and Javanese is high.

There are two chief varieties of hepatic carcinoma. The hepatoma or liver cell type is much the more common. The other is the cholangioma or bile duct type. On clinical grounds there is something to be said for the separation of a special type of cancer with cirrhosis. It is generally held that cirrhosis of the liver is a precancerous condition. This has been discussed in these pages on several occasions. A high incidence of cirrhosis is shown in cases of primary cancer. Multilobular or portal cirrhosis occurs in middle or later life, almost invariably after the age of thirty, and practically every patient with primary carcinoma is over the age of thirty. There is much evidence to indicate that most hepatic carcinomata (especially hepatomata) arise in cirrhotic livers. The incidence of cirrhosis is definitely greater in hepatoma than in cholangioma. Carcinoma in a cirrhotic liver is the terminal phase of definitely understood pathological changes. Portal cirrhosis and acute cytolytic necrosis are due to the action of toxic substances definitely known (trinitrotoluene) or strongly suspected (alcohol or some associated substance). Cytolytic necrosis occurs rapidly and the end result depends on the severity of the intoxication together with the extent of the destructive lesion. When necrosis is very extensive death from acute yellow atrophy occurs within a few days. If a small part of the liver withstands the toxic attack, the patient may survive for weeks or months, the destroyed hepatic tissue being replaced by cellular fibroblastic tissue and the persisting areas of parenchyma being in a condition of more or less active proliferation, the condition being subacute atrophy or necrosis. Some may survive for months or years. In these what has been destroyed may be compensated for, to some extent, by regenerated liver tissue. These livers may display multiple nodular hyperplasia, with nodules of functioning regenerated liver tissue separated by areas of post-necrotic fibrosis. In cytolytic necrosis the whole damage is done at one blow. The lesion becomes static when the destroyed tissue is fibrosed and some regenerative hyperplasia supervenes. In cirrhosis the toxic action is long maintained. The destructive effect probably lasts many years, with necessity for compensatory hypertrophy. The lesion is progressive and does not always stop at multiple nodular hyperplasia. There are cases in which liver cell adenomata form, which may be followed by malignant disease. C. Saburin suggested that mul-

[1] *The Lancet*, September 12, 1931.

tiple nodular hyperplasia may be intermediate between cirrhosis and carcinoma. Stewart believes that this applies only to the nodular hyperplasia of cirrhosis. In those who recover from cytolytic necrosis and in whom the regenerated liver suffices to preserve life, there is, he states, no further destruction of cells and no further demand for compensatory hypertrophy occurs. Cirrhosis exhibits progressive cell destruction followed by compensatory hypertrophy and hyperplasia which, in some advanced cases, goes beyond the normal and assumes the autonomous character of a neoplasm.

In discussing the unicentric or multicentric origin of liver cell cancer, Stewart points out that liver adenomata are often multiple, particularly in cirrhosis, but that the seemingly multicentric cancer can be explained. Primary cancer, especially of liver cell type, readily invades the portal vein and extends in both directions along the lumen, forming a tree-like cast. The growth-distended vessels, on section, have the aspect of a multicentric tumour. Dissemination readily occurs. Invasion of the hepatic vein is less common and the inferior *vena cava* is rarely permeated by growth. Bile duct cancer is more liable than liver cell cancer to cause extrahepatic metastases, but less often grossly invades the portal and hepatic veins.

The ætiology of hæmochromatosis is obscure and in Stewart's opinion no definite conclusion is yet possible. In many instances of multilobular cirrhosis there is a varying degree of siderosis of the liver cells, but there is no evidence that any close relation exists between the two conditions. However, in the *post mortem* room hæmochromatosis is always associated with some hepatic cirrhosis and might be expected to have some association with primary cancer of the liver. In a series of autopsies, in 15 of 151 cases of hæmochromatosis, primary cancer of the liver was present. In cirrhosis generally the percentage of primary cancer was 3·41 or, excluding the slighter degrees of cirrhosis, 7·3%. Significance, however, cannot be attached to the apparently higher cancer rate in hæmochromatosis. Cirrhosis is the causative factor and the siderosis probably plays no direct part. In hæmochromatosis primary cancer of the liver resembles the disease as it occurs in ordinary portal cirrhosis.

Professor Stewart makes no mention of syphilis as a possible determining factor in malignant degeneration of the liver. We know, however, that syphilitic lesions may closely simulate either primary hepatic cancer or secondary metastasis from elsewhere, and differential diagnosis may be extremely difficult. Considerable cachexia may attend the syphilitic lesion. In some tropical countries primary hepatic carcinoma seems to be common. In the tropics also the liver is very prone to suffer from other disorders. The presence of biliary helminthiasis may be a factor in determining cancer. *Schistosoma japonicum*, clonorchis and ascaris infectations as well as dicrocœliasis may be ætiological agents. The supervention on cirrhosis is significant. As regards secondary cancer of the liver following primary growth in the colon or rectum, the bromsulphalein test of hepatic efficiency may save the patient a futile operation.

GASTRO-JEJUNO-COLIC FISTULA.

N. HAMILTON FAIRLEY, who has carried out a great deal of investigation in sprue, has, in company with T. Pomfret Kilner, reported three cases of gastro-jejuno-colic fistula with megalocytic anæmia simulating sprue.[1] They point out that gastro-jejuno-colic fistula is not very rare. In 1925 Verbrugge collected records of 202 cases from the literature and added 14 from the Mayo Clinic. In these reports anæmia *per se* did not attract special attention, and it is from this point of view that the present report is made. In all three cases the clinical picture resembled that of sprue, and in all there was an anæmia of the megalocytic type. In two of the cases the gastric secretion of hydrochloric acid was high, and in both there was a temporary response to liver extract given in adequate dosage. Analysis of the fæces revealed a high fæcal fat content with adequate splitting. The blood calcium content was lowered and this lowering was associated with a normal inorganic phosphorus and a low serum bilirubin content. Two of the patients had been subjected to gastro-enterostomy, and the third had a carcinoma involving the stomach, jejunum and colon. Fairley and Kilner state that defective absorption of essential foodstuffs following short-circuiting of the small intestine is obviously the basis of certain clinical features in gastro-jejuno-colic fistula and that it probably accounts for the close clinical and biochemical similarity to sprue. Sprue is essentially a disease of the gastro-intestine, characterized by defective absorption of fat, calcium and possibly of glucose. In sprue there is deficient production of hydrochloric acid in the stomach. Fairley and Kilner also refer to the absence in sprue of the stimulating factor shown by Castle to be necessary for the normal erythroblastic activity of the bone marrow. In the absence of this factor megaloblastic hypertrophy of the marrow ensues and megalocytic anæmia is produced. It is important to note that in the first two cases fractional test meals revealed high acid curves and that there was a lack of the satisfactory therapeutic response to high protein, low fat and low carbohydrate dietary seen in sprue.

These cases are of the greatest interest, particularly in regard to the causation of megalocytic anæmia. Fairley and Kilner attach much importance to malabsorption by the intestine and to the absence of the "specific factor". It would be interesting to know what importance, if any, they attach to the infection of the upper part of the alimentary tract by organisms from the colon. They emphasize an important clinical point that intractable diarrhœa following gastro-jejunostomy should be regarded as originating from a gastro-jejuno-colic fistula until such a diagnosis is disproved.

[1] *The Lancet*, December 19, 1931.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Biotropism.

J. H. SEQUEIRA (*The British Journal of Dermatology and Syphilis*, November, 1931) discusses the explanation of various unusual phenomena which are the subject of the book "*Le Biotropisme*", by Milian. Milian's observations led him to the hypothesis that many substances have the property of exalting the vitality of parasites of the human body; to this faculty he applies the term "biotropism". Examples are given of many instances in which the introduction of vaccines, sera and chemicals into the body, increases the virulence of microbes. It is shown that cases of furunculosis, herpes and acute rheumatism frequently lie dormant; syphilis is another outstanding example of latency. Various streptococcal infections and malaria are often brought to light by the administration of a drug. The administration of a vaccine of the organisms of typhoid and paratyphoid *A* and *B*, injections of milk, infectious fevers, have stimulated a latent syphilitic focus and produced a reaction to the Wassermann test. The author claims that this stimulation occurs more commonly with the arsenical compounds, and that the phenomena occur especially when the medication is inadequate; also that some of the conditions attributed to anaphylaxis or the destruction or diminution of immunity are really due to the stimulation of the microbe, and disappear with increased doses. It is held that doses sublethal to spironemata may not only stimulate the organism, but eventually lead to the development of arsenic-resistant strains, and there is often a grave danger of activating latent syphilis by insufficient arsenical treatment. It is suggested that arsenic, bismuth and mercury are desirable in the treatment of syphilis.

Jugular Compression in Neuro-Syphilis.

D. C. SMITH AND J. A. WADDELL (*Archives of Dermatology and Syphilology*, November, 1931) observed that jugular compression increased the intracranial pressure and the rate of flow of spinal fluid. Hence they decided to combine the use of jugular compression with the administration of "Neoarsphenamine". The cases were selected, as in certain instances this procedure would obviously be contraindicated. Twenty-two patients were treated by this method; there were eighteen control cases. Compression was applied for thirty minutes following the injection of the arsenical preparation, and a quantitative determination of the arsenic content of the spinal fluid was made by the Gutzeit method. The results were tabulated. The average arsenic content of the spinal fluid in the control cases was 0·002 milligramme per five cubic centimetres. Two of the controls did not react to the test. In the cases of jugular compression the average content was 0·0031 milligramme per five cubic centimetres, an increase of 55%. There was a marked variation in the arsenic content in both the compression and the control cases. This method is suggested as a possible adjunct in treatment.

Fatal Iodine Dermatitis.

J. J. ELLER AND E. C. FOX (*Archives of Dermatology and Syphilology*, November, 1931) report a fatal case of iodine dermatitis which presented many unusual features. The eruption consisted of macules, papules, tubercles, and rupoid, fungating and granulomatous ulcerations. Four months after the eruption (which followed a few weeks of medication with potassium iodide) appeared, the patient died of profound toxæmia. The patient passed large quantities of iodides in the urine. Iodides were also found in the skin, liver and kidneys *post mortem*. There was a great diversity of opinion among the pathologists as to whether the lesions were neoplastic or inflammatory. It is suggested that, following the ingestion of an iodized salt for some years, the patient in the case reported was sensitized to such an extent as to develop a fatal dermatitis after the ingestion of a tonic containing small amounts of potassium iodide.

Rhus Toxicodendron Dermatitis.

E. GRIMES (*Archives of Dermatology and Syphilology*, November, 1931) reports the disappearance of an extensive poison ivy dermatitis within thirty-six hours of a motor accident, in which the patient suffered considerable extravasation of blood. Acting upon this result, twenty other patients were treated in a similar manner by the injection of quantities of their own blood. In all cases the condition was cured in from one to four days. In some a second injection of whole blood was given intramuscularly at the end of forty-eight hours.

Alepol in Leprosy.

B. B. DIKSHIT (*The Indian Medical Gazette*, January, 1932) discusses the employment of "Alepol" in the treatment of leprosy. "Alepol" was evolved through the desirability for a less irritating substance than the hydnocarpus derivatives usually employed. It is a sodium salt of certain fatty acids of *Hydnocarpus wightiana* that have a comparatively low melting point. The author has treated 200 patients by means of intramuscular and intravenous injections of "Alepol" without the use of any other directly anti-leprosy drugs. The dose is regulated according to the condition of the individual patient. As a rule, the commencing dose employed by the author was one cubic centimetre of a 3% solution, given intramuscularly. Injections are given twice a week, and the dose increased on each occasion by 0·5 cubic centimetre until a maximum of five cubic centimetres is reached. From this point it is found most convenient to administer the drug intravenously. A dose of one cubic centimetre of a 1% solution is given at the commencement; the dosage is then gradually increased until it reaches five cubic centimetres. The strength of the solution is then increased, and the quantity decreased to one cubic centimetre, as before. This is gradually increased until, in favourable cases, nine or ten cubic centimetres of a 2% solution may be given. For intravenous administration the drug is dissolved in Locke's solution without glucose. The patient's tolerance of the dose may be tested by means of the erythrocyte sedimentation test. If the difference between the patient's erythrocyte sedimentation index before and twenty-four hours after injection is less than ten, the dose is not too great and can safely be increased at the next injection. If the difference is greater than ten, the dosage is too great. The results of treatment in the author's series were satisfactory, and compared very favourably with the results of treatment with hydnocarpus oil. "Alepol" has a distinct advantage over the other salts of hydnocarpus oil in that it is less irritating in its local action.

Lupus Erythematosis Disseminatus.

W. H. MOOK, R. S. WEISS AND L. K. BROMBERG (*Archives of Dermatology and Syphilology*, November, 1931) describe thirteen cases of generalized *lupus erythematosis*. Clinical evidence of tuberculosis was found in only four of the cases. In each instance this evidence was strikingly meagre, and there was no sign of active tuberculosis. The authors are inclined to agree with those who contend that *lupus erythematosis* is a toxic or septic cutaneous symptom. They regard leucopenia as an important symptom. Attention is called to the frequent involvement of the serous cavities. In six of the nine fatal cases the duration of the disease was less than one year. The other three patients lived from two and a half to six years.

UROLOGY.

Cancer of the Bladder.

M. R. DARGET (*Journal d'Urologie*, February, 1931) pins his faith to radium needles applied through the open bladder in the treatment of carcinoma of that organ. In contradistinction to most French urologists, he has found the results to be most encouraging. He complains that his compatriots have insisted on burying this method before it has been given a fair chance to live. In his 23 cases proved to be cancer by histological examination, the results have been

excellent in nine and good in four. This result is claimed to be extremely satisfactory in the present state of knowledge. In five cases the condition of the bladder was such that any method was foredoomed to failure, and in the light of his experience the author would not employ radium were he treating these patients today. Five patients died within a month or two of the operation, death being due to pulmonary or cerebral trouble and probably hastened by the intervention. The author deprecates any method of applying radium other than by open exposure through a suprapubic incision, and the needles, which contain three milligrammes of radium element, filtered by 0·5 milligramme of platinum, must be so inserted and retained that they do not become detached. They are deeply implanted in wheel-spoke fashion, at intervals of 1·5 centimetres, entering at the periphery of the tumour. The author places a little balloon filled with about 70 cubic centimetres of solution, in the bladder, so as to exert pressure on the tumour and so keep the needles in place, as well as to form a protective screen for the healthy mucosa. When all the conditions of operation have been favourable so that the limits of the tumour have been well seen and the needles satisfactorily implanted, the needles are left in for five days, but the irradiation must be prolonged to seven days if the technique has been imperfect. Cystoscopy must follow the operation every three months for eighteen months and thereafter every six months. A definite relapse calls for a first application of radium by the same technique, though it will be more difficult, since the exposure will be restricted.

Pyelography with "Abrodil".

J. VON HERMAN AND A. MELLY (*Zeitschrift für Urologie*, Volume XXV, Number 8, 1931) express great satisfaction with the use of "Abrodil" for intravenous pyelography. It possesses an advantage over "Uroselectan" in that there is no uncomfortable general effect on the patient during the injection. The solution can be filtered before it is boiled. It is crystal-clear in appearance and produces no tissue reaction if a small quantity of it is accidentally injected outside the vein. The specific gravity of the urine is very little raised by the injection. "Abrodil" can therefore be given to patients who are even in a preanæmic state. With good renal function the excretion is quick and the optimum time for the first exposure is from five to fifteen minutes after the injection.

Dynamism in Urology.

F. LEGUEU (*Journal d'Urologie*, January, 1931) discusses the ideas developed in his clinic in Paris in connexion with the importance of dynamic factors in obstruction or stasis in the urinary tract. The earliest clinical observers in the field of urology knew that the mere mechanical factors of obstruction by a stone, an enlarged gland or a stricture, did not account for all the phenomena present. Dynamic factors were superadded, and they were of great importance; they depended upon muscular, and particularly upon neuro-muscular disturbances. With the ureter it is the same as with the more adequately observed lower urinary tract; the upper end of the ureter and the pyelo-ureteral junction are the domain of very intense recent interest. Such neuro-muscular disturbances of the pelvis and upper part of the ureter have been suspected, but their actual presence was not brought to light until the introduction of pyeloscopy in Legueu's clinic. That these pyeloscopic appearances are real is shown by applying serial pyelography as well. Legueu looks forward to the day when cinematography of the movements seen during pyeloscopic examination will become a really practicable method. Only recently it has been shown that the dilatation which always occurs above the level of suture of a divided ureter is not dependent on a narrowing of the suture area, but on functional peristaltic trouble, with incoordination of contractions leading to atony of the ureter. There are several main types of functional disturbance of the renal pelvis: (i) spasms, leading to contraction of the pelvis, holding it in a fixed attitude for some time and preventing evacuation; (ii) slow evacuation, due to infrequent, weak and lazy contractions, even when stimulated by the injection of hypertonic solutions; (iii) incontinence, when the complex sphincters in the pyelo-ureteral area seem to exert no control, but allow the pelvis to be evacuated in a continuous fashion; (iv) retentions, purely functional, with no mechanical obstruction present, incomplete or complete, all leading to greater or less distension of the pelvis and calyces. It is important that one should not simply cure the mechanical trouble, as, for instance, performing nephropexy to straighten a kinked ureter; it is necessary to see whether a functional disturbance is also present. The rational treatment for such neuromuscular disturbances is only possible if the fundamental determining cause of such disturbance is known. Such cause may be hormonic or even toxic, or it may arise locally from external compression or internal obstruction, or be simply chemical, from some irritative quality of the urine. But if all these causes be reasonably excluded, it is found that the origin lies in the nervous system itself, and here the various denervation operations find their place in therapy.

Pararectal Prostatectomy.

GIL VERNET (*Journal d'Urologie*, May, 1931) presented a new method of perineal prostatectomy before the French Urological Society. It is called pararectal prostatectomy. Briefly, his technique is as follows: (i) A four centimetres skin incision is made parallel to and one centimetre from the top of the anus. (ii) The ano bulbar raphé is retracted posteriorly and divided. (iii) With a finger in the rectum the posterior part of the mid-point of the perineum is divided, the lax cellular tissue between the circular and longitudinal layers of rectal muscle being entered (that is, behind the *recto-urethralis* muscle). (iv) These layers are separated until a retractor can be inserted. Then a bloodless gauze dissection is continued until the prostate is palpated anteriorly. (v) A two centimetres incision is made in the posterior surface of the prostate to expose the prostatic urethra. (vi) The retractor is withdrawn, the urethra being torn with the finger and enucleation being completed, as in the suprapubic route operation. (vii) The retractor is reinserted and the cavity examined for "tags and bleeders". (viii) A de Pezzer catheter is inserted and packing is used. (ix) Two skin sutures only are used. Post-operative care consists in daily dressings and antiseptic lavage. The de Pezzer catheter is removed on the second day and an indwelling catheter inserted through the penis; for this it may be necessary to use a finger in the wound as a guide. The catheter is removed on the tenth to twelfth day and the patient discharged by the fifteenth day. The author claims for this operation simplicity, absence of trauma, a bloodless field and rapid and efficient functional results. There are 80 cases in his series and no mortality is mentioned. He states that any patient who can stand suprapubic cystostomy, is suitable, and two-stage operations are unnecessary.

Retention of Urine.

M. BETTANCOURT (*Journal d'Urologie*, August, 1931) describes three cases of acute retention of urine in which immediate relief was obtained by the urethral injection of a 2% solution of "Stovain". The patients were men, the eldest being forty-five years old, and retention was apparently due to inflammation superadded to urethral stricture or calculus.

Gonorrhœa.

FOR the treatment of acute and chronic gonorrhœal infections, B. Ferguson (*Urologic and Cutaneous Review*, August, 1931) advocates the simple stimulation of an artificial leucocytosis with a minimum of local treatment. His system consists in a triweekly intravenous injection. Two of the injections consist of 15 cubic centimetres of a one in 1,500 solution of hydrochloric acid in distilled water; the third is three cubic centimetres of "colloidal mercury sulphide-Hille". These injections are found to induce a marked but transitory increase in the leucocytes of the peripheral blood. The author claims highly satisfactory results and quotes several illustrative cases of cure after the failure of recognized methods of treatment. No statistics are given.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on October 2, 1931, DR. F. HOPE MICHÔD, the President, in the chair.

Cardiac Arrhythmia.

DR. ELLIS MURPHY read a paper entitled: "Cardiac Arrhythmias" (see page 283).

DR. S. F. MCDONALD said he had listened to Dr. Murphy's paper with great interest and had learned a great deal from it. He thought that in all these arrhythmias the electrocardiographic curve was useful to a great extent, but most practitioners did not visualize the curves of the electrocardiogram as the actual movements of the heart, as they did with the signs recorded by trained eyes, ears and fingers. If one were taught to set the curves by the corresponding pulse and stethoscopic records, one could follow them.

With regard to the patient with coronary thrombosis mentioned by Dr. Murphy, there had been no classical symptoms at all, merely dyspnœa on walking up hill, and a very slow pulse, varying from 42 to 46 per minute. Listening to this patient's heart, one heard a systolic murmur, then an abrupt second sound with a diastolic murmur immediately following, then a pause, then the systolic murmur again, and then the second sound. There was a series of extrasystoles, not sufficient to produce a second sound. If this record had been put alongside the curve of the electrocardiograph, it would have been appreciated much better. Lewis used this method.

With very few exceptions one should be able to recognize arrhythmia by the ordinary clinical methods—by eyes, ears and fingers, and especially by fingers.

Dr. McDonald wished to ask whether strophanthus or other preparations had replaced digitalis to any extent or whether the old preparations were best. He agreed with Dr. Markwell that the digitalis leaf was much the best, particularly for patients taking small doses frequently. These people were much more pleased to have a tablet to swallow unobtrusively instead of drops to pour out, and "Nativelle" granules were very valuable for this reason.

The question arose of how far the clinical side of paroxysmal tachycardia and flutter was likely to influence the general outlook of the patient's life. This arose in regard to life insurance policies. Should the history of an attack be sufficient to prevent the acceptance of a life insurance policy?

Had Dr. Murphy seen very much of *pulsus alternans*, which Mackenzie and Lewis so feared and distrusted? Were the modern views the same as Mackenzie's?

DR. C. A. THELANDER asked what was the bearing of these tachycardias on surgery, particularly in exophthalmic goître. Could any definite limit be stated? Some years ago, before the preparatory iodine treatment had come much into use, he had two patients urgently requiring treatment. In one case the pulse could not be counted, but the heart's action was quite regular at 240 per minute before and during operation. Under local anæsthesia both superior thyreoid arteries were ligated, and about a week later a lobe was removed, and the patient made an excellent recovery. The other patient had a rapid and irregular heart, and had been refused operation, on account of the heart condition, by several prominent men in the south. Here also a two-stage operation was quite successful.

The preliminary iodine treatment was most important, but should be regarded only as the first stage of surgical treatment. He had seen a number of cases in which ambulatory patients had been taking iodine for many weeks; the results were sometimes disastrous, for, if tachycardia recurred, it was very difficult to treat.

Dr. Murphy, in reply to Dr. McDonald, said that there was an instrument which represented the heart sounds and worked in conjunction with the electrocardiogram; Lewis illustrated this. Dr. Murphy did not think that any other drug could replace digitalis as a routine. In acute cases strophanthin, given intravenously, or ouabaïn was good, but digitalis was the sheet anchor in ventricular tachycardia. In myocardial degeneration or coronary disease digitalis did not help very much; there was no such dramatic effect as when rapid heart beats were present. One of the best methods of giving digitalis was by the leaves, though one was so used to giving the tincture that a mental jolt was necessary. With regard to insurance and the tachycardias, Dr. Murphy thought the verdict would depend on the condition of the cardiac muscle. The very name "paroxysmal" meant that it only lasted a short time. A clinical examination of the heart should be made in the period between the attacks of the tachycardia, and an electrocardiogram taken; and if this were satisfactory, the patient was a "normal risk", for a person could be subject to paroxysmal tachycardia and yet live to a ripe old age. The condition of the heart muscle was the main thing. Hume Turnbull, in Melbourne, put forward the idea that fibrillation and tachycardia were functional conditions. *Pulsus alternans* was now generally not considered as serious, as Mackenzie had said. Frequently Mackenzie would give a prognosis of death in six months. Very frequently there was myocardial damage and heart bundle block, and though these were of grave significance and associated with a moderate degree of cardiac damage, they were not so bad as was previously considered.

In reply to Dr. Thelander, Dr. Murphy said that cardiac arrhythmias in relation to exophthalmic goître formed a very interesting subject. He thought a heart rate of 240 a minute would be very difficult to count. He felt he could not say enough with regard to the preoperative treatment of exophthalmic goître. He considered the patient should be given digitalis first, then the date of the operation should be fixed, and ten days previous to this date Lugol's iodine treatment should be commenced. Dr. Murphy said that since he had been following this routine he had had more success than previously. Another most important thing was that if the patient developed a thyreoid storm on the morning of the operation, operation must be postponed and done later. With regard to fibrillation in goître, this in many cases would be fibrillation in a fairly small heart, and suitable for treatment with quinidine, but it tended to recur if quinidine were given before operation. The better plan was to remove the goître and then give quinidine, and one was much more likely to get a good result.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Lawrance, Kenneth George, M.B., 1929 (Univ. Sydney), Coast Hospital, Little Bay.

Sampson, Bernard Moore, L.R.C.P., 1903 (London), M.R.C.S., 1903 (England), Rydalmere Mental Hospital.

Tannahill, Robert William, M.B., 1931 (Univ. Sydney), "Eastwell", Cammeray Avenue, North Sydney.

Ward, John Thomas, M.B., B.S., 1929 (Univ. Sydney), 7, Moonbie Street, Summer Hill.

Warden, Douglas Arthur, M.B., 1930 (Univ. Sydney), "Tabourie", Muston Street, Mosman.

Correspondence.

DIET DEFICIENCY AND TRACHOMA.

SIR: In his letter to THE MEDICAL JOURNAL OF AUSTRALIA published in the issue of October 10, to which attention

has been drawn in the current number of *The British Journal of Ophthalmology*, Dr. Lockhart Gibson refers with kindness to a paper of mine on the epidemiology of trachoma (*The British Journal of Ophthalmology*, July, 1931). He comments on the absence of mention of a deficiency of vitamin *A* in the diet as a contributory agent in the causation of trachoma.

It is usually accepted that any depressed physical condition presents a more favourable culture medium for the infective principle of a contagious disease than perfect health. It may be conceded, therefore, that this may be the case with trachomatous infiltration of the eye and its appendages. But trachoma is constantly being seen in children who have enjoyed the rich and varied diet which is common in the homes of well-to-do fellaheen in Egypt, as well as in the homes of Europeans, and who are apparently in admirable physical condition.

In exceptionally severe cases of trachoma which may be met with in stages Tr. IIa and Tr. IIb, it is common to find a severely depressed physical condition due to famine, ankylostomiasis, bilharziasis, or all these conditions combined.

I must therefore state that as far as my experience goes, neither defective diet nor a depressed physical condition (including the so-called adenoidism) is an essential factor in the process of trachomatous infection.

Dr. Lockhart Gibson is not quite correct in stating that primary infection with trachoma was unknown in Egypt during the war. Colonel Eason, A.M.S., reported that in two and a half years between 1916 and 1918, out of 30,000 eye cases among the troops he found 63 men to have contracted trachoma in Egypt or Palestine (*Bulletin of the Ophthalmological Society of Egypt*, 1919).

I agree with Dr. Lockhart Gibson that there is no reason why a patient with certain stages of trachoma, such as Tr. III, should not occupy a bed in the general ward of a hospital, provided that: (i) ordinary hospital regulations are observed, including a special towel for each patient; (ii) the ward is staffed by adequately trained nurses; (iii) flies are excluded from the ward or are present sparsely; (iv) neither the trachoma patient nor the other patients in the ward have any definite conjunctival discharge.

In the last paragraph of the editorial note in *The British Journal of Ophthalmology*, January, 1932, it is suggested that an experimental study on the question of diet as an essential factor in infection with trachoma would not present insuperable difficulties. Unfortunately, it has not been conclusively proved that any living creature, other than man, can either naturally or by inoculation be made to exhibit those clinical signs by which the disease is recognized.

Yours, etc.,

A. F. MACCALLAN.

London,
January 7, 1932.

TRAUMA AND ORGANIC VISCERAL DISEASE.

SIR: In a recent paper (see THE MEDICAL JOURNAL OF AUSTRALIA, November 7, 1931) I mentioned that in certain medico-legal cases there quoted, medical witnesses had stated that muscular effort caused increased peristalsis of the intestine. I did not reveal the names of these learned gentlemen. In both cases, as anyone can verify—and as I have done—the thesis was put forward without condition or qualification, as a general statement. I offer no apology for recording this statement. I had a good right to do so, and exercised my right reasonably.

But it annoyed Dr. F. S. Stuckey. Resenting the appearance of a sceptic, he has had the nerve to "protest" against the recording of a statement of general application and, if true, of great importance, on the ground that a particular case was in view at the time, although any logical mind can see that it cannot matter whether the immediate occasion was an alleged trauma to a human being, or whether it was some laboratory experiment in which the subject was Dog 1, Cat 2, or Rabbit 3.

That was protest number 1. Then he went on superbly: "I wish . . . also to protest against the gibe at the opinions of other medical men without producing any evidence to show their falsity." He said "opinions", but what I recorded was a single opinion, however shared. Had I wished, I could have pointed to my second paper (of same date) as ample evidence.

Now he comes along with a second letter. We rub our eyes. He tells us that his previous letter "was in no sense a defence of the thesis that muscular effort may increase peristaltic action". He would now "welcome a reference to any authoritative standard or discussion of this question". He wants it to be "authoritative". He has already had, I think, a reasonable discussion in my second paper, but we can let that go. And he asks then if it may not be "still another of those many queries to which we answer 'we do not know'".

And that is what has become of the thesis, "Lifting or any exertion would increase peristalsis" (wording duly verified) which Dr. Stuckey claimed that I had to prove false.

So there you are. What can anybody do with a controversialist like that?

Finally, readers will now give Dr. Stuckey all the sympathy they consider due in respect to his twice-repeated grouch that there is one misguided court in Australia that does not think it enough just to wait and hear what that gentleman thinks, and register judgement accordingly.

Yours, etc.,

C. E. CORLETTE.

January 23, 1932.

STERILIZATION OF THE UNFIT.

SIR: On reading Dr. Merrillees's letter in your issue of the twenty-third instant in reply to my comments on the above subject I realize nothing would be gained and much of your valuable space taken up in a useless controversy with this gentleman; however, I am obliged to reply to his queries for enlightenment. Far from being "a fuss", I regard it as a duty and a privilege to comment upon any policy advocated by the medical Press of Australia that may have far reaching effects upon society generally and with which the prestige of the medical profession is so intimately involved. If Dr. Merrillees will again refer to my letter he will see therein the reasons why I sought permission to designate as atrocious the suggestion of the sterilization of patients with mental disease as a condition to their discharge from hospital, and I am sure he does not wish me to repeat myself here. In addition, he will receive further enlightenment by referring again to Dr. Ellery's letter, the latter dealing more fully with this aspect of the subject. I am sad to hear, assuming it to be true, that it is known to all physicians in general practice that the prohibition of the marriage of defectives leads to masturbation, abortion, infanticide, and all sorts of psychopathic relations between males, and it would be sadder still to contemplate these same physicians advising marriage as a means of combating these psychopathic behaviour anomalies, and it appears the doctor has placed the cart before the horse in this connexion. The facts supporting the assertion that mental defect is due to genetic variation and the heterozygous nature of its hereditary transmission may be found in a study of the essential psychopathic features in the histories of a sufficient number of cases, together with the facts gleaned by experienced workers in this field of research over a great many years.

I have already endeavoured to show in my letter that sterilization, regarded from the viewpoint of practical preventive medicine in practice, would be practically negligible, so why the necessity of useless repetition? As regards the doctor's hypothetical case, I think he would be doing a duty to his patient as well as to society and humanity generally if he took steps to see that such an individual was placed in such circumstances where he or she would receive adequate and proper supervision which the history of the case reveals to be sadly lacking, and I can assure him that the modern care and treatment of

such cases does not involve solitary confinement or any other imaginary gruesome method, and I have no hesitation in claiming segregation to be the only satisfactory solution in such a case, for, no doubt, otherwise "the good work would go on"—with venereal disease in its train, even though the social code might be appeased by the absence of the fœtus.

Dr. Merrillees appears to have been so confounded by my use of the term "atrocious" that he has made confusion worse confounded. If he will refer again to my letter, he will find I sought permission to apply this term to the suggestion that sterilization be made a condition to the discharge of patients from hospital, an aspect of the subject with which he has not dealt.

Yours, etc.,

GREY EWAN.

Mental Hospital,
Newcastle,
January 25, 1932.

COMPARISON OF THE KLINE AND WASSERMANN TESTS.

SIR: In today's issue of the Journal "Practitioner", commenting on a recent comparison of the Kline and Wassermann tests, inquires wherein lies the advantage of testing sera by more than one method.

As one who has worked with Kline and in fact brought his test over to the Newcastle Hospital in 1928, where it has been used routinely in conjunction with the Wassermann test ever since, I may be permitted to stress some of the advantages we have experienced through the use of the dual test.

First, the Kline test has been found more sensitive than the Wassermann test in early syphilis, and also in cases of treated syphilis. It is a common occurrence to find a patient with a history of recent contact with the disease, who gives a positive Kline and negative Wassermann. Here our procedure is to go right ahead with intensive treatment and wait a few weeks for a second and possibly confirmatory Wassermann. It must be remembered that a positive Wassermann, in this country, is the commonly accepted medico-legal proof of syphilis. Hence, where the Kline is positive and the Wassermann is negative, we hesitate to label a patient officially as a syphilitic. Our faith in a positive Kline test, however, is so complete that we regard each patient with one as being in need of antispecific therapy.

Secondly, with the Kline test, all sera tested give definite results, whereas only ninety-five Wassermanns out of every hundred are satisfactory, the remainder being anti-complementary.

Thirdly, the Kline test has the advantage over the Kahn and Wassermann tests in that it is much simpler and takes much less time to carry out. Several Newcastle practitioners are now employing the test in their private practices because it is handy, efficient and convenient. In cases where a patient gives a negative Kline, they are reasonably well satisfied to dismiss the possibility of syphilis. On the other hand, with a positive Kline appearing, the patient is usually sent along to a pathologist for a confirmatory Wassermann. This system, apart from the saving of time, is often a saving in pathologist's fees to the patient who is free from syphilis, but is having a blood test as part of a routine examination.

Variation in results of the two tests, which occurs in from five to eight *per centum* of cases, must, of course, be interpreted in the light of the clinical facts and the history of the individual case.

A study of Kline's protocols published in the *American Journal of Syphilis* (Volume X, Number 4, October, 1926, page 636), in which are set out comparisons of 2,800 tests, will show how this is done and will answer "Practitioner's" final query regarding the reporting and interpreting of results.

I am convinced that in the near future the Kline, or microscopic slide precipitation test, as its originator prefers it to be called, will gradually replace the Wassermann altogether, but until it is sufficiently well known in Australia as a very definite and efficient test for syphilis, it is better for the present to couple it with the conventional and better known Wassermann.

Yours, etc.,

THOMAS HAMILTON.

Newcastle,
February 6, 1932.

SIR: In your issue of February 6 "Practitioner" asked for amplification of the final point in our summary: "It is an advantage to test a serum by more than one method."

In the report of the Laboratory Conference on the Sero-Diagnosis of Syphilis, convened at Montevideo, September 1930, by the Health Organization of the League of Nations, the conclusions arrived at were a reaffirmation of those obtained from the previous conference, held at Copenhagen, 1928. On page 18 we read: "Even most sensitive methods have resulted, in certain cases, in negative reactions where methods otherwise less sensitive have given positive; it was therefore urged that two different diagnostic methods should be employed simultaneously. Thus a greater guarantee is obtained against possible errors, miscalculations *et cetera*." From the work described in these reports it is evident that no method is capable of giving 100% of positive results in known cases of syphilis.

The Kline test has only recently come into use, and has not yet been compared on a large scale with other standard methods; but by virtue of its simplicity it will inevitably appeal to serologists. If further experience supports the claim that a Kline reaction appears earlier than a Wassermann, then the clinician will be able at an earlier date to proceed with treatment with greater confidence.

With regard to "Practitioner's" concluding question: What explanation is given when reporting conflicting results obtained by different methods? A serologist's report is purely informative. The present knowledge of colloids is insufficient to enable us to give an adequate explanation of occasional discrepancies. We should expect that a practitioner receiving an equivocal report would consider the case worthy of further serological investigation, both immediately and after an interval. It is the practitioner's responsibility to decide whether treatment be administered during that interval.

It is perhaps needless to add that in our series those sera giving conflicting results were retested, and, where possible, further tests were carried out on other samples of serum from the same cases to confirm the report.

Yours, etc.,

PHYLLIS G. ASHWORTH.
DOROTHY H. IRVING.

Melbourne,
February 10, 1932.

DIATHERMY OF TONSILS.

SIR: In a previous letter I questioned whether any of the exponents of diathermy of tonsils would have the courage to ask their colleagues to bring forward through these columns their unsatisfactory cases. I suggested that if they did so, they would be overwhelmed. To my knowledge, no such request has been issued.

Now, in your issue of February 13, 1932, Dr. Kent Hughes says that for some years he has asked for reports of unfavourable cases, and no one has come forward with any. I wish to be convinced about his results, but I would suggest that colleagues would be very diffident of bringing forward in these columns Dr. Kent Hughes's unsuccessful or unsatisfactory cases unless he makes a very distinct request that they do so.

I now make the remarks in my previous letter more personal by asking Dr. Kent Hughes whether he has the courage to ask his colleagues to bring to light in these columns his unsatisfactory cases, if they exist.

Yours, etc.,

A. B. K. WATKINS.

Newcastle,
February 13, 1932.

THE CASINO FRIENDLY SOCIETIES HOSPITAL.

SIR: It has been brought to the notice of the Committee of the North-Eastern Medical Association that the management of Friendly Societies' Hospital at Casino has invited applications for a medical practitioner to take charge of that hospital.

In view of the history of the hospital, and in view of certain statements made by members of the management, my Committee would request that intending applicants communicate with the Medical Secretary, New South Wales Branch, British Medical Association, or with the Honorary Secretary, North-Eastern Medical Association, Lismore.

Yours, etc.,

J. R. RYAN,
Honorary Secretary, North-Eastern Medical Association.

Lismore,
February 16, 1932.

THE WORSHIP OF THE TEST TUBE.

SIR: Dr. Ellery must be congratulated not merely for his views on test tube worship, but for the fine literary flavour of their expression. It is with great regret, however, that we cannot agree with his delightful theory of the trance. Not even prolonged worshipping at the shrine of glassware could produce ecstasy of years' duration. As a matter of fact, in these times of uncertainty such a state would be highly prized, and if anyone could produce a recipe for its production, many would be grateful for its publication.

But returning from literature to science, surely we are at cross purposes, since I certainly agree with Dr. Ellery that:

(1) Students should have a sound psychological training.

(2) That it is possible to fall too early into the habit of laboratory investigation of neurotics.

(3) That there is some truth in Edward Strecker's dictum. Furthermore, I would add that so far as it is possible to assess percentages, in the aggregate, 70% of all medical treatment is psychological.

The test tube method is not dangerous *per se*, provided that the clinician appreciates the psychological factors involved in testing. Admittedly, the indiscriminate use of test tube methods is reprehensible, and any procedure undermining the faith in the physician and diverting attention from the correct diagnosis must inevitably assist the quack.

The point which I wished to raise was precisely that of the psychology of the test tube. So long as its cost is excessive, automatically it is given an exaggerated place in the armoury. The patient's attention is riveted to the test as a species of grand consultation, whereas its real significance is of no more value than an estimation of pulse rate. To give an example: If nature had provided the human race with a large abdominal window opening directly into the stomach, it is certain that to raise the flap and to view the stomach would be a routine procedure in every case showing gastric symptoms. No great psychological principle would be concerned in this simple thirty second operation. Unfortunately for us, to acquire even a modicum of this information, a lengthy, complex and expensive procedure is involved. In order to obviate the psychological result, simplification is necessary, and one means of doing this is by reducing the cost.

Whether under ideal conditions, which undoubtedly include a clinician trained in psychology, test tube methods would be used more or less frequently, is a matter of conjecture. Personally, I am of the former persuasion; but time will show. As neither Dr. Ellery nor myself is likely to see the medical millennium, we must leave the final answer to the next generation.

Yours, etc.,

JOHN BOSTOCK.

Wickham Terrace,
Brisbane,
February 8, 1932.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on February 1, 1932.

The Senate resolved to place on record its appreciation of the long and faithful service rendered to the University and to the Faculty of Medicine by the late Dr. Robert Henry Todd during his occupancy of the Lectureships in Medical Jurisprudence and Medical Ethics.

It was resolved to convey grateful thanks to Mrs. Gordon Craig for the handsome gift of a book plate for the 677 volumes comprising the medical library presented to the University by the late Dr. Gordon Craig.

On the recommendation of the Conjoint Board, the following appointments to the medical staff of the Royal Alexandra Hospital for Children were approved:

Lecturer in Medical Diseases of Children: Dr. E. H. M. Stephen.
Lecturer in Surgical Diseases of Children: Dr. R. B. Wade.
Honorary Surgeon: Dr. W. Vickers.
Honorary Physician: Dr. M. J. Plomley.
Honorary Assistant Physician: Dr. L. H. Hughes.
Honorary Ear, Nose and Throat Surgeons: Dr. W. A. Dunn and Dr. H. Huff Johnston.
Honorary Assistant Ear, Nose and Throat Surgeon: Dr. O. A. Davey.
Honorary Assistant Pathologist: Dr. W. K. Inglis.
Honorary Assistant Relieving Surgeon: Dr. T. Y. Nelson.

The following Examiners were appointed for the Final Degree Examination in Medicine:

Medicine: Dr. A. E. Mills, Dr. S. A. Smith.
Clinical Medicine: Dr. E. W. Fairfax, Dr. A. W. Holmes à Court.
Surgery: Professor H. R. Dew, Dr. E. M. Fisher.
Clinical Surgery: Dr. A. Aspinall, Dr. J. L. McKelvey.
Obstetrics: Professor J. C. Windeyer, Dr. P. L. Hipsley.
Clinical Obstetrics: Dr. Constance D'Arcy, Dr. A. J. Gibson.
Gynæcology: Dr. R. L. Davies, Dr. Cedric Bowker.

THE WILLIAM GIBSON RESEARCH SCHOLARSHIP FOR MEDICAL WOMEN.

MISS MAUD MARGARET GIBSON has placed in the hands of the Royal Society of Medicine a sum of money sufficient to provide a scholarship, of the yearly value of £292, in memory of her father, the late Mr. William Gibson, of Melbourne, Australia. The scholarship is awarded from time to time by the Society to qualified medical women who are subjects of the British Empire; and is tenable for a period of two years; but the period may in special circumstances be extended to a third year. The next award will be made in June, 1932.

In choosing a scholar, the Society will be guided in its choice "either by research work already done by her, or by research work which she contemplates. The scholar shall be free to travel at her own will for the purpose of the research she has undertaken".

There is no competitive examination, nor need a thesis or other work for publication or otherwise, be submitted. The Society has power at any time to terminate the grant if it has reason to be dissatisfied with the work or conduct of the scholar.

Applications should be accompanied by a statement of professional training, degrees or diplomas, and of appointments, together with a schedule of the proposed research. Applications must be accompanied by testimonials, one as to academical or professional status, and one as to general character. Envelopes containing applications *et cetera* should be marked at the top left-hand corner, "William Gibson Research Scholarship", and should be addressed to Mr. G. R. Edwards, Secretary, Royal Society of Medicine, 1, Wimpole Street, London, W.1. They should be in the Secretary's hands by Wednesday, June 1, 1932.

NOTICE.

A DEMONSTRATION will be held at the Psychiatric Clinic, Broughton Hall, Wharf Road, Leichhardt, on Thursday, March 3, 1932, at 2.30 p.m. Patients suffering from the following conditions will be shown: Recurrent ataxia, apoplectiform confusion, myelitis, spondylitic muscular atrophy, Parkinson's syndrome (post-encephalitic), presbyophrenia with chorea, and progressive muscular atrophy.

Diary for the Month.

MAR. 1.—New South Wales Branch, B.M.A.: Ethics Committee.
MAR. 3.—Victorian Branch, B.M.A.: Branch.
MAR. 3.—South Australian Branch, B.M.A.: Council.
MAR. 4.—Queensland Branch, B.M.A.: Clinical Meeting.
MAR. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. R. J. Wheeler has been appointed Junior Resident Medical Officer at the Perth Hospital, Western Australia.

* * *

Dr. A. Williams has been appointed Medical Officer of Health by the Denmark Road Board, Western Australia.

* * *

Dr. J. N. H. Wilson has been appointed Government Medical Officer at Adaminaby, New South Wales.

* * *

Dr. J. M. Main has been appointed Government Medical Officer at Barellan, New South Wales.

* * *

Dr. F. W. Fay (B.M.A.) has been appointed a member of the Hobart Public Hospital Board, Tasmania.

* * *

Dr. A. M. Cudmore (B.M.A.) and Dr. P. S. Messent (B.M.A.) have been appointed members of the Dental Board of South Australia, under the provisions of the *Dentists' Act*, 1931.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser" page xiii.

ADELAIDE CHILDREN'S HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Honorary Officers.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Ophthalmologist.

MATER CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: House Physician, House Surgeon.

SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Assistant Radiologist.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Relieving Assistant Aural Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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